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TISSUE CHANGES IN SINUS MEMBRANES AFTER PENICILLIN TREATMENT

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After six years of work on the histopathology of experimentally produced sinusitis, we were convinced by 1935 that "owing to the defensive factors inherent in sinus epithelium and the connective tissue elements of its tunica propria, almost every sort of surface application to such membranes becomes an irritant unless its strength be isotonic."[1] This point was amplified in our discussion of the defense mechanisms of the upper respiratory tract in 1937.[2]

Among the various chemotherapeutic agents (zephiran, parachlorophenol, and tyrothricin) recently tested for use in wound healing, Howes[3] found that dilutions strong enough to inhibit bacterial growth were toxic to epithelial cells and fibroblasts. Streptomycin and sulfamylon were mildly inhibitory of cell growth. However, penicillin calcium proved inert as to cellular damage.

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Read before the American Laryngological Association, St. Louis, Mo., April 24, 1947.

Numerous reports of excellent results from the local use of penicillin, as nose drops, by the Proetz postural method, by instillation through cannulas, and by atomization, have been current for the past few years. It has become fairly evident, through many clinical trials, that local use of this valuable antibiotic is only justified in the very early stages of sinus involvement; that certain bacterial types are penicillin-resistant, while others may become so; that some individuals are allergic to penicillin; and that chronic infections are unlikely to be helped by such local applications.[4-6] Our own experience for the past three years has convinced us that penicillin used locally in the sinuses is of value only in acute and subacute cases.

No reports of microscopic examination of acutely inflamed sinus membranes have come to our attention. It was therefore decided to infect a number of frontal sinuses of cats with a virulent hemolytic streptococcus culture of human upper respiratory origin, using the techniques described in our reports of 1931 and 1932.[7, 8]

Six cats were inoculated with beta streptococcus pyogenes, grown in a Hartley broth culture and introduced by puncture into each right frontal sinus on November 6, 1946. On November 8, the left frontal sinuses were similarly inoculated. On November 11, all of the sinuses of four of the animals were perfused with sodium penicillin at a concentration of 2,000 units per cc. of physiological sodium chloride solution, about 0.5 cc. of the solution being injected into each sinus. The animals were sacrificed (or died) on the dates indicated in the individual protocols. The frontal and nasal bones were dissected out, the bone above the sinuses was chipped away, and the sinus membranes were fixed, in situ, in formol-Zenker solution. After the usual subsequent preparation for histologic study, paraffin sections were stained with hematoxylin-eosin-azur or with Goodpasture's stain for bacteria in tissues.

The sections show the following features:

CAT 1.—Weight 5 lbs. Tissues secured November 15, four days after use of penicillin.

Left sinus. The membrane is greatly thickened, showing an acute stage of reaction to infection (as shown in earlier experiments[1, 2]), and modified by a large number of macrophages and plasma cells in addition to the predominant polymorphonuclears characteristic of acute reaction. The epithelium is broken in many places,

but elsewhere shows ciliated columnar cells and goblet cells. No granules are seen in the epithelium or elsewhere.

Right sinus. The membrane is thickened and filled with polymorphonuclears, plasma cells and histiocytes. There is a larger proportion of plasma cells than in the left sinus membrane; no granules are seen.

CAT 2.—Weight 4½ lbs. Tissues secured November 14.

Left sinus. The membrane is edematous and thickened and filled with many polymorphonuclears, plasma cells and histiocytes. The epithelium shows numerous purple-like granules (streptococci), especially in the goblet cells but also between the columnar cells; many are dumbbell shaped.

Right sinus. The membrane is thickened and filled with numerous polymorphonuclears. A deep pocket of streptococci (Goodpasture stain) surrounded by a dense mass of cells occurs below a broken part of the epithelial surface. No granules appear on the epithelial surface.

CAT 3.—Weight 4½ lbs. Tissues secured November 13.

Left sinus. There is marked acute inflammation. Polymorphonuclears and macrophages are predominant.

Right sinus. There is subacute inflammation. The membrane is thinner than on the left side and with fewer polymorphonuclears. Goblet cells contain numerous purple-blue granules resembling streptococci.

CAT 4.—Weight 4½ lbs. Animal died November 8, six hours after the second inoculation with streptococcus culture. No penicillin treatment. Sinus membranes fixed immediately.

Left sinus. The membrane shows an early stage of inflammation. Some polymorphonuclears are present, and the membrane is beginning to thicken. The goblet cells are laden with streptococcus-like granules; there are also some in the columnar cells of the epithelium.

Right sinus. The membrane shows an early stage of inflammation. Streptococcus-like granules are seen on the epithelial surface and in the epithelium. There are rounded masses of granules in the epithelium and in the goblet cells. Some plasma cells are present in the fibrous layer of the membrane.

CAT 5.—Weight 4½ lbs. Tissues secured November 12.

Left sinus. The membrane is too broken for good sections, but apparently normal.

Right sinus. The membrane shows an early stage of inflammation, with fairly numerous polymorphonuclears and many macrophages with granules; there is also engorgement of the blood vessels.

CAT 6.—Weight 4½ lbs. Animal died during night of November 8, following the second (left) inoculation with streptococcus culture. No penicillin treatment.

Left sinus. The membrane is thin; no indication as yet of inflammation.

Right sinus. There is acute inflammation, with many polymorphonuclears, histiocytes, etc., and thickening of the membrane. Polymorphonuclears are also seen in the epithelium. Goodpasture stain shows cocci in many cells.

Comment. The acute inflammatory reaction appears to continue longer after local treatment with penicillin than in the earlier experiments in which no such treatment was employed. This suggests that the active stage of phagocytosis by polymorphonuclears is of longer duration and is, therefore, in all probability more effective. Some allege that penicillin in solution is a local cellular irritant exciting polymorphonuclear activity, but it may possibly exercise a true bacteriostatic stimulus to phagocytosis, at least in living ciliated membranes.

In the two animals that received no penicillin, and in a deep mucosal pocket of one so treated, streptococcic granules were present. Similar granules were found in the goblet cells of some of the other animals, but none on the surface. Thus it would seem that in the cat streptococci are rapidly destroyed on the surface and in the epithelium of acutely inflamed sinus membranes through the bacteriostatic properties of sodium penicillin, 2,000 units per cc. of physiological sodium chloride solution.

For perfusion of acutely inflamed sinuses in human subjects and for intratracheal injection, we have found during the past three years that a solution containing 1,000 units per cc. gives rise to no complaint of local burning or irritation. This adds strength to the hypothesis that phagocytosis is greatly accelerated by the local use of penicillin.

MEDICAL ARTS BUILDING.

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XXVII

TRACHEOTOMY IN BULBAR POLIOMYELITIS

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This paper summarizes experiences with tracheotomy in patients with bulbar poliomyelitis treated at the University of Minnesota Hospitals and the Minneapolis General Hospital during the 1946 epidemic. During 1946, 1830 cases of poliomyelitis were treated in Minneapolis. Almost all occurred during the epidemic period. Approximately 400 were diagnosed as "bulbar" cases. Tracheotomies were performed in 75 of these. Cases were classified as bulbar when the pharyngeal and laryngeal nerve supply was involved and also when there was circulatory and respiratory failure pointing to disease of the bulbar centers controlling these functions. In many patients spinal components were also present and in some encephalitic involvement was suggested. Because poliomyelitis is a contagious disease, the patients were treated in only a few centers having isolation facilities.

Prior to this epidemic the staff members of the various departments involved had had no experience with tracheotomy in poliomyelitis. Using the few published reports as a guide tracheotomy was employed cautiously. As the epidemic progressed the indications became clearer. We hope that a summary of our experiences may be useful to others.

From the Division of Otolaryngology, University of Minnesota Medical School, and the Otolaryngology Services of the University of Minnesota Hospitals, and the Minneapolis General Hospital.

Aided by a grant from the National Foundation for Infantile Paralysis, Inc.

Read at the Middle Section Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Chicago, Ill., January 20, 1947.

As a result of our experience we believe that tracheotomy has an important place in the treatment of bulbar poliomyelitis. In certain patients we think it is a life-saving procedure. There is reluctance on the part of some physicians to accept tracheotomy as a useful adjunct in the treatment of poliomyelitis. Any physician whose experience with bulbar poliomyelitis encompasses only a few cases may question the need for tracheotomy. However, an epidemic in which several hundred bulbar cases occur produces a considerable number which meet the criteria for tracheotomy outlined in this paper.

In a thorough search of the literature from 1900 through 1946 we found no report of a large group of poliomyelitis patients subjected to tracheotomy. No further reports were found in a bibliography covering the literature from 1789 to 1944.[13] We suspect that the procedure has been carried out many times without being reported. The background literature will be briefly reviewed.

Galloway[1] reported three patients who had respiratory difficulty as a complication of poliomyelitis. In two of his patients he believed tracheotomy was a life-saving factor. In one patient Galloway observed a pool of fluid in the pharynx with its gravity level in the larynx and trachea. In his other cases thick mucus was aspirated when the trachea was opened.

In a comprehensive review of poliomyelitis Davison[2] states that tracheotomy may be necessary in bulbar poliomyelitis.

Wilson,[10] writing from the Infants' and Children's Hospital in Boston and the Department of Pediatrics of the Harvard Medical School in 1931, succinctly expressed the considerations passing through the mind of the physician contemplating tracheotomy in poliomyelitis patients. He stated:

"During the past year, however, we have encountered several situations where in spite of all care we have felt it necessary to take further steps and have resorted to tracheotomy. Ideally, if a tracheotomy is to be performed, it should be done before aspiration of foreign material has taken place and before choking attacks occur. Actually, however, it seems impossible to determine ahead of time which patients are going to need tracheotomy. We realize that this is a radical procedure, that the majority of patients with pharyngeal paralysis develop no such serious situation, and that unquestionably many patients who have aspirated foreign matter into the bronchial tree, who have been subject to severe choking attacks and have become dangerously fatigued, have survived without any such operation. We have performed tracheotomy in a few cases, however, when the

need has already developed, and obtained good results. Tracheotomy enables the patient to be fed with much less fear of choking attacks. It is quite easy to keep the trachea itself free from secretions by direct aspiration through the tracheotomy tube. Twice when an emergency tracheotomy has been performed on children who collapsed after choking attacks, a large amount of mucus has been removed directly from the trachea with dramatic relief of symptoms. We feel that this procedure offers considerable assistance in a few selected cases."

About ten years later Wilson[3, 7] further emphasized his continued belief that occasionally tracheotomy is a life-saving procedure.

Glaser[4] describes the use of a positive pressure oxygen system connected to the patient's trachea by a tracheotomy tube. He reports one recovery and one death with his method.

Nelson-Jones and Williams[5] reported the case of a dental officer with dyspnea due to pharyngeal and laryngeal paralysis due to poliomyelitis. Pharyngeal secretions were spilling into the trachea. Tracheotomy provided immediate relief of the laryngeal stridor. Aspiration cleared the bronchi of secretions and the patient ultimately recovered.

Durand[6] emphasized that drowning rather than paralysis causes death in many bulbar cases. He cited recoveries after placing nearly moribund patients so that their heads were low and their faces were down to permit gravity drainage of pharyngeal secretions.

Our Criteria for Tracheotomy in Bulbar Poliomyelitis. In discussing the criteria for tracheotomy used in the 1946 Minneapolis epidemic, two groups of patients must be considered. One group includes patients over 14 years of age admitted to the neurology service of the University Hospitals. The second group includes pediatric patients at the University Hospitals and all poliomyelitis patients at the Minneapolis General Hospital. Criteria for tracheotomy for these two groups of patients were not identical.

On the adult neurology service at the University Hospitals we were requested to do a tracheotomy on any patient whose bulbar symptoms were early and whose disease was progressing rapidly, or who had evidence of involvement of the circulatory or respiratory centers, or who had severe toxicity or mental changes.

On the University pediatric service and on the contagious service at the Minneapolis General Hospital (both adults and children), the

patient with bulbar poliomyelitis who had pharyngeal paralysis severe enough to allow spillage of pharyngeal secretion into the larynx and trachea was subjected to tracheotomy only when he could not clear his own airway or when his airway could not be cleared for him by pharyngeal aspiration and postural drainage.

Inability of the patient to clear his own airway was indicated by recurrent cyanosis, coarse, bubbling râles in the chest, laryngeal stridor and inability to cough efficiently. These symptoms are most important in excited patients who are unmanageable and in stuporous patients. Direct laryngoscopy may demonstrate pooling of mucus in the pharynx, paralysis of one or both cords, and diminished tactile sensitivity of the laryngeal mucosa.

The second group of criteria discussed above apply when one does not feel justified in performing the operation prophylactically. We are not entirely satisfied that delaying the operation until positive indications exist is always best. However, the judgment of the physician enters the problem at this point and we feel certain that most tracheotomies in poliomyelitis will continue to be done only when definite indications exist. We feel strongly that a new concept as to what constitutes proper indications for tracheotomy in bulbar poliomyelitis should be created in the minds of physicians. We hope that this discussion will help to delineate this concept.

The use of the respirator in poliomyelitis patients having both bulbar and spinal cord lesions is contraindicated according to several writers because the machine may suck pharyngeal secretions down into the lower respiratory tract. Tracheotomy has been used in several of our cases to permit aspiration of this material from the trachea and bronchi.

Pathological Physiology. Aspiration bronchitis and pneumonitis, anoxia and fatigue are the conditions to be minimized by keeping the airway clear. We have had several experiences which have demonstrated to our satisfaction that gross amounts of pharyngeal secretion and food do enter the trachea and cannot be evacuated by the patient. Upon opening the trachea in one of our early patients thick gray liquid welled up into the wound. After this had been aspirated and the operation completed the wound filled again. The patient's anoxia decreased and his condition improved immediately when his airway was finally cleared. Similar conditions have occurred 5 times in 22 tracheotomies done at the Minneapolis General

Hospital and 10 times in 53 tracheotomies done at the University Hospital.

When the patient with bulbar poliomyelitis coughs or is cyanotic after eating or drinking, the entrance of food into the respiratory tract must be suspected. This was dramatically illustrated in a seven-year-old girl whose eating was observed six weeks after tracheotomy. This child's operation had been done because of midline flaccid vocal cord paralysis, laryngeal stridor and recurrent anoxia. The patient also had had the usual bulbar symptoms of nasal speech and dysphagia. At the time the child's eating was observed she swallowed well considering the extent of her original pharyngeal paralysis. She was drinking chocolate milk when we watched her. Milk spilled over into her airway. She coughed, and the chocolate stained the dressing around her tracheotomy tube. Such spillage occurred with each feeding. It is hard to believe this child could have escaped serious pulmonary involvement during six weeks of laryngeal paralysis without artificial tracheal aspiration. Tracheal aspiration could only be provided in such a case by tracheotomy or repeated bronchoscopy. Such bronchoscopy is obviously not practical. It should be emphasized that according to competent pediatric opinion there is almost 100 per cent recovery of function of paralyzed pharyngeal muscles if life can be preserved in bulbar poliomyelitis through the first week of the disease.[3]

Certain defense mechanisms which either normally bar the entrance of foreign substances into the trachea or aid in the eviction of such material are rendered impotent in some cases of poliomyelitis. The defenses in question are: normal vocal cord movement and normal sensitivity of the laryngeal mucosa.

Motor paralysis of the larynx was observed in 12 cases. Intralaryngeal anesthesia was demonstrated in one case. These conditions may have existed undiagnosed in other cases inasmuch as we did not freely employ direct laryngoscopy or bronchoscopy as a diagnostic measure in patients in very poor condition. If knowledge of the exact state of the larynx is to be had, laryngoscopy is necessary.

When the motor innervation of the larynx has been damaged, the exact position of the paralyzed cords varies. This is what one would expect because of variability in the destruction of the cells of the motor nucleus (nucleus ambiguus) which govern abduction and adduction. Seven patients had bilateral paralysis with the cords nearly in contact in the midline on inspiration. The cords were blown apart on expiration. Three patients had total paralysis

of one cord. This cord lay in the midline. One of these patients could abduct the other cord only feebly. Two other patients had weak adductor muscles on one side and therefore coughed inefficiently.

Coughing is normally carried out by bringing the vocal cords together in the midline, building up positive intrathoracic pressure, suddenly abducting the vocal cords and allowing the expiratory air blast to carry foreign material out of the trachea. Patients having weakened or paralyzed cords cough inefficiently. Decreased sensitivity or total anesthesia of the larynx superimposed on an already damaged cough mechanism greatly increases the vulnerability of the trachea. We have encountered intralaryngeal anesthesia of a degree sufficient to permit direct laryngoscopy and bronchoscopy of conscious patients with little discomfort. Laryngeal anesthesia in poliomyelitis has been described by others.[8]

Patients having bilateral flaccid cord paralyses are unable to breathe deeply because their cords are pulled together by air entering the lungs during inspiration. As the cords are pushed downward by the inspiratory air stream, the glottic opening is narrowed. Continued breathing through a narrowed glottis is possible but difficult. Such breathing must be pathologically shallow to prevent asphyxia, because each really deep inspiration shuts off the airway. Such a fine degree of respiratory control is difficult or impossible in the excited, weakened patient with a damaged respiratory mechanism. Entrance of secretion into such a larynx is disastrous.

Transient anoxia is common in bulbar poliomyelitis patients. The importance of anoxia has recently been emphasized by Galloway.[9] Only a brief discussion will be given here. Of all body tissues, neural tissue is the least capable of withstanding oxygen want.[11] This is especially true in poliomyelitis where the central nervous system is already damaged by the virus. The primary purpose of tracheotomy is to insure proper and constant oxygen supply. Weakness, fatigue, accumulation of secretion in the airways, and the toxicity resulting from the illness combine to hinder proper aeration of blood with resultant central nervous system anoxia. Just how much anoxia a particular patient's already damaged brain can tolerate is hard to determine. At the University of Minnesota Hospitals an oximeter employing photoelectric cells fastened to the ear lobe was employed to determine the degree of blood oxygenation. The usual clinical signs of oxygen want occur only when anoxia is already rather far advanced.

Early in anoxia combativeness resembling that of acute alcoholism may be present. Other symptoms include confusion, disorientation, irrationality and coma. Failure to recognize anoxia as a possible cause of these symptoms, and assumption that the polio-encephalitis per se is the cause, may misdirect therapy. The administration of sedatives to such patients can be dangerous. Efforts to insure adequate oxygenation of the central nervous system often produce great improvement.

We have observed children who responded well and appeared not to be seriously ill at the onset of an episode of respiratory obstruction. These children have then become cyanotic and have never recovered consciousness although a few have lived for several days. If tracheotomy would avoid even one of these severe anoxic periods in such cases, it would seem to be worthwhile. The margin between life and death in patients gravely ill with poliomyelitis is so narrow that one anoxic episode may kill them.

On the basis of their experience on the adult neurology service at the University Hospitals Brown and Baker[12] believe that from a treatment standpoint bulbar poliomyelitis can be divided into two large groups. In the first, the predominant lesions occur in the region of the cranial nerve nuclei; while in the second, the involvement is localized in the reticular substance which contains the autonomic centers for respiration and circulation. The former is the most commonly recognized type and is generally the one diagnosed clinically. With the exception of the glossopharyngeal and vagus nerves, paralysis of the cranial nerves alone does not present any real danger to life.

Brown and Baker state further that when the involvement of the brain stem is more extensive and implicates not only the cranial nerve nuclei but also the respiratory regulating centers, the outlook is more grave. These cases can be detected chiefly by the symptoms referable to a failure of the respiratory center. These symptoms generally develop somewhat later in the illness than do the cranial nerve palsies and manifest themselves by an irregular rhythm and depth of respiration with a tendency to shallowness and prolonged intervals between inspirations. These pass on to prolonged periods of apnea and finally cessation of the respirations. Some of the patients may show evidence of a circulatory collapse with a dusky red, florid appearance, a rapid irregular pulse, and a low blood pressure that is often difficult to obtain or maintain. The most extensive lesions in these individuals are found in the reticular substance of

TABLE 1.—NUMERICAL DATA PERTAINING TO TRACHEOTOMIZED BULBAR POLIOMYELITIS PATIENTS.

	MINNEAPOLIS GENERAL HOSP.	UNIVERSITY HOSPITAL NEUROLOGY SERVICE	UNIVERSITY HOSPITAL PEDIATRIC SERVICE	TOTAL
Number of tracheotomies	22	35	18	75
Survived	7	13	9	29
Died	15	22*	9	46
Lived because of tracheotomy***	4	6**	7**	17
Probably lived because of tracheotomy	1	0	1	2
Prophylactic tracheotomy with recovery	2	7	1	10

*Brown and Baker[12] believe that most of these deaths occurred in patients having respiratory and circulatory center damage and that barring accidental mechanical respiratory obstruction (e.g., aspiration of vomitus or plugging of tracheal cannula by secretion) patients having primary cranial nerve nuclear damage lived if proper oxygenation of their central nervous systems was maintained. The first four adults having "pure" cranial nerve nuclear involvement died without tracheotomy. After tracheotomy was used in these cases accidental respiratory obstruction accounted for their only deaths, according to Brown and Baker.

**Positive pressure vests, Drinker-type respirators, and photoelectric oximeters were used in these University Hospital cases together with tracheotomy. Omission of any one of these factors might have resulted in death. All respirator patients are now living outside the machines.

***For detailed case studies see report of cases below.

the medulla, destroying the autonomic centers for control of respiration and circulation. If the damage to these areas is extreme, the outcome is fatal in spite of all efforts to maintain these patients. On the other hand, many patients develop only a mild or moderate involvement of the autonomic centers and can be maintained by elective tracheotomy and adequate oxygenation. However, in such patients even a short period of obstruction or cyanosis may result in irreversible damage to the nervous system with a rapid downhill course.

Numerical Data. Numerical data pertaining to 75 tracheotomized patients are presented in Table 1. We emphasize that this table

represents a lumping of the data on all the patients who had tracheotomies whether they had only cranial nerve nuclear involvement or that plus respiratory and circulatory center involvement, as outlined above in the discussion of the paper of Brown and Baker.

The information shown in Table I does not lend itself to statistical analysis because of the small number of cases. Furthermore it seems unlikely that sufficient data ever will be accumulated to be statistically significant because few epidemics are larger than the one reported here. Patients with the bulbar type represent a small proportion of the total; the number of these patients requiring tracheotomy is still smaller. Collection of statistics from several epidemics is a possible means of securing information but variations in handling of patients make such comparisons inaccurate.

The writers have been exceedingly conservative in constructing Table 1. When a patient is listed as having "lived because of tracheotomy," the evidence has been very conclusive. Most patients listed as having "probably lived because of tracheotomy" might properly be listed as having actually lived because of tracheotomy. Patients listed as having "prophylactic tracheotomies" were operated upon because they were unable to swallow and it seemed that their paralysis was progressing. Obviously an unknown proportion of this group would have entered one of the more dangerous classifications without tracheotomy, and also some would have recovered.

In no case did we feel that tracheotomy caused death. Complications of tracheotomy included mediastinal emphysema, subcutaneous emphysema and tracheitis. Of the 29 patients who survived 27 have now been decannulated. Two still wear tubes. One adult has become psychologically dependent on her tube and the other, a child of 3 years, has residual laryngeal paralysis.

Unsuccessful Methods of Aspiration. We tried various types of continuous and intermittent pharyngeal suction using inlying catheters inserted through the nose. These catheters had multiple openings to prevent obstruction. Continuous suction in our experience has not been satisfactory except in a few instances. If one could use a closed system such as the urologist or the thoracic surgeon employs, continuous suction might be satisfactory. In the open pharynx one must move the catheter around to reach the secretion. We have found that the small, admirably silent, Stedman pump develops insufficient negative pressure for withdrawal of thick pharyngeal secretions. The usual water suction is extremely noisy and may be dangerous. We had one fatal accident when the water backed

up and passed into a child's pharynx through the pharyngeal suction catheter. We finally came to use intermittent nasopharyngeal suction with the conventional type of portable electric pump despite the noise. Sometimes a nasal catheter was left in place and was moved about when aspiration was carried out. Leaving the tube in place spared the patient the discomfort of having it passed repeatedly.

The question of intubation with O'Dwyer tubes instead of tracheotomy came up. In addition to the ever-present possibility that the O'Dwyer tube may be coughed up one cannot aspirate the trachea through this tube. The tube provides an open channel for entrance of pharyngeal secretions into the trachea. Furthermore one is not dealing with a stenotic larynx through which he wishes to establish an air channel. The O'Dwyer tube would provide a narrower airway than is already present. Because the larynx is not stenotic, the tube would not remain in place even as well as in acute inflammatory laryngitis. Furthermore laryngeal spasm would ensue in some patients. Consideration of these factors led to rejection of the O'Dwyer tube without trial at the Minneapolis General Hospital. It was actually used once at the University Hospital but was unsatisfactory.

The endotracheal tube used for anesthesia has also been rejected. Such tubes are practical if one is sure that they will be needed for only a short time. It would be impossible to leave such tubes in place for weeks while awaiting recovery from pharyngeal paralysis. These tubes were employed to maintain an airway while tracheotomy was being done.

SUMMARY AND CONCLUSIONS

Experiences with tracheotomy during the Minneapolis poliomyelitis epidemic of 1946 are analyzed. 1830 cases of poliomyelitis were treated in Minneapolis during 1946. Approximately 400 "bulbar" cases were reported. Seventy-five tracheotomies were done. Twenty-nine of these patients survived.

The tracheotomies were done at the Minneapolis General Hospital and the University of Minnesota Hospitals. On the University Hospitals' pediatric service and at the Minneapolis General Hospital, tracheotomy was performed when the following indications existed: respiratory distress as evidenced by recurrent cyanosis, coarse râles in the chest and laryngeal stridor; excitement and unmanageability causing the patient to resist pharyngeal aspiration; stupor of a degree sufficient to make the patient oblivious of accumulation of

secretion in his airway; inability to cough effectively; pharyngeal pooling of mucus, vocal cord paralysis, or intralaryngeal hypesthesia demonstrable by laryngoscopy.

On the adult neurology service at the University Hospitals the criteria listed above were considered to be indications for tracheotomy. The physicians in charge of the University adult neurology service came to believe that tracheotomy gave the patient his best chance if done before cyanosis occurred, because cyanosis can only be diagnosed clinically when it is rather far advanced and when anoxia has done irreversible damage to a central nervous system already markedly damaged by the virus.

The detrimental effect of anoxia on the central nervous system is emphasized. We believe that this has been stressed sufficiently by only a few authors writing about bulbar poliomyelitis. Numerical data are condensed in Table 1.

As a result of our experience in this epidemic we believe: (1) that tracheotomy improves the chance for survival of properly selected bulbar poliomyelitis patients, if done before anoxia has produced significant central nervous system damage; (2) that tracheotomy used in conjunction with various means for producing artificial respiration will enable some critically ill poliomyelitis patients to survive until natural recovery of damaged neural tissue can occur.

REPORT OF CASES

The 17 cases listed in Table 1 as having "lived because of tracheotomy" are summarized below to permit the reader to judge the propriety of their inclusion in this group. Complete case summaries are not given; only information pertaining to the subject of this paper is given. All cases were typical bulbar poliomyelitis with characteristic history, physical and laboratory findings.

CASE 1.—J. B., M. G. H. No. 5466A, a 7-year-old female, was cyanotic and showed suprasternal and ensiform retraction when excited. Direct laryngoscopy showed total midline paralysis of the right vocal cord and partial paralysis of the left cord. After tracheotomy total inability to swallow continued for three and a half weeks. The patient then partially recovered the swallowing function but laryngeal paralysis persisted for three weeks more. Part of all food swallowed entered the trachea and had to be aspirated through a tracheal cannula. Her condition did not permit extubation until two months after tracheotomy.

CASE 2.—P. H., M. G. H. No. 5734A, an 8-year-old male, had a history of swallowing difficulty with much mucus in his throat for two days before admission. One severe cyanotic episode occurred in the hospital about six hours after admission. A tracheotomy was done. No further cyanosis occurred despite extreme

restlessness with disorientation, hyper-pyrexia, and combativeness for four days after tracheotomy. The swallowing function had returned 11 days after tracheotomy. The tube was removed 18 days after the operation.

CASE 3.—G. S., M.G.H., No. 7108A, a 30-year-old male, had moderate cyanosis on admission; there was no dysphagia or respiratory difficulty but he had noted a slight change in his voice since the onset of his illness. Breath sounds were absent over the right chest and there were coarse ronchi over the left lung. A tracheotomy was done one hour after admission and much thick mucoid material was aspirated from the tracheobronchial tree. Color and breath sounds became normal. Thereafter atelectasis recurred in the right lung whenever suctioning was inefficiently done. The patient survived paralytic ileus and was extubated 18 days after tracheotomy.

CASE 4.—D. L., M. G. H. No. 7045A, a 16-year-old male, had dysphagia and an accumulation of much pharyngeal mucus for two days following admission. He became combative and disorientated and would not permit pharyngeal aspiration. A tracheotomy was done to prevent his choking on pharyngeal mucus. He was disorientated and very ill, with circulatory and respiratory irregularities for three days after tracheotomy. The tube was removed 18 days after tracheotomy.

CASE 5.—D. F., U. H. No. 769133, a 6-year-old male, had difficulty in swallowing, "bulbar speech," mental cloudiness, and irregular respirations. A tracheotomy was done because this bulbar poliomyelitis patient was to be put into a respirator and a means of tracheal aspiration had to be provided. Following tracheotomy the patient was in the respirator 17 days, then was extubated.

CASE 6.—P. K., U. H. No. 769051, an 8-year-old male, was lethargic and there was much accumulated pharyngeal mucus. As the trachea was opened secretion welled up into the incision and much mucus was aspirated from the tracheobronchial tree. Once during operation the child stopped breathing. The patient was placed in a respirator five hours after tracheotomy and removed therefrom only after two months and was extubated thereafter. X-ray films showed pneumonic and atelectatic changes in the lungs throughout these two months.

CASE 7.—E. D., U. H. No. 769394, a 16-year-old male, was mildly cyanotic, and there was an accumulation of pharyngeal mucus before tracheotomy. He was disorientated and had Cheyne-Stokes breathing for several days after tracheotomy. A respirator jacket and a helium-oxygen mixture were used for a few days. The patient was extubated 20 days after tracheotomy.

CASE 8.—E. A., U. H. No. 769422, a 29-year-old male, was disorientated, had difficulty in swallowing and was about to enter the respirator because of intercostal paralysis when a tracheotomy was done. Positive pressure respiratory equipment was used. The patient was extubated 14 days after tracheotomy. This patient is included in the group "saved by tracheotomy" because he was a bulbar poliomyelitis patient on whom a mechanical respiratory apparatus had to be used.

CASE 9.—M. K., U. H. No. 769563, a 14-year-old female, was cyanotic, had mottled skin and weak pulse for four hours before tracheotomy. She was disorientated and unresponsive, and there was an accumulation of mucus in the pharynx. She had a series of convulsions after tracheotomy. These were controlled by pentothal and curare. She was extubated 12 days after tracheotomy.

CASE 10.—D. T., U. H. No. 769527, a 13-year-old male, had nasal speech, swallowing difficulty, and vocal cord paresis suggested by aphonia. From the

third to the eighth postoperative day respirations were very irregular with apnea and cyanosis occurring, so that a respirator was used for varying lengths of time. The patient was extubated 33 days after tracheotomy.

CASE 11.—E. M., U. H. No. 769570, a 25-year-old male, with dysphagia and progressing bulbar involvement was unable to swallow for six weeks. His temperature was markedly elevated for 10 days. The patient was extubated after seven weeks.

CASE 12.—A. P., U. H. No. 769956, a 31-year-old male, had dysphagia, and excess mucus in the pharynx; and was disorientated. There was bilateral ptosis and external ophthalmoplegia. The patient was in a positive pressure respiratory apparatus and was extubated 23 days after tracheotomy.

CASE 13.—R. B., U. H. No. 771118, a 2-year-old male, had excess mucus in the pharynx and the tracheobronchial tree (aspirated at the time of the tracheotomy), and was disorientated. The patient became cyanotic on the way to the operating room. He was semicomatose for three days after tracheotomy. A respirator was used. The patient has not been able to be extubated to date (six months later).

CASE 14.—N. M., U. H. No. 771242, a 33-year-old male, had dysphagia, bulbar speech, and cyanosis except when receiving oxygen before tracheotomy. The patient was extubated 25 days after the operation.

CASE 15.—M. A., U. H. No. 771705, a 28-year-old female, was extremely ill, and was placed in a respirator a few hours after tracheotomy which was done on admission. The patient had frequent febrile episodes, and massive atelectasis of the left lung on one occasion. She has resisted extubation because she is psychologically dependent on the tube.

CASE 16.—M. C., U. H. No. 771985, a 9-year-old male, was disorientated, had an excessive amount of pharyngeal mucus at the time of tracheotomy. The patient was obviously destined to be placed in a respirator at the time of tracheotomy. An x-ray film showed atelectasis of the right upper lobe two weeks after tracheotomy. The patient was extubated but the exact time was not recorded.

CASE 17.—G. B., U. H. No. 771600, a 5-year-old male, resisted aspiration of the pharynx by the nurses, was unable to cough, and had many râles in his chest prior to tracheotomy. Excess mucus was aspirated when the trachea was opened. Irregular respirations became regular following tracheotomy. The patient was extubated but the exact time was not recorded.

We thank Dr. Irvine McQuarrie, Professor and Head of the Department of Pediatrics and Dr. A. B. Baker, Associate Professor and Director of the Division of Neurology, for their comments, criticisms and suggestions concerning the portions of the paper dealing with material from their departments.

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XXVIII

HEARING IMPAIRMENT DUE TO CRANIOCEREBRAL TRAUMA

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The hearing function can be damaged by an injury to the head. The impact may be by way of the face, the vault or the vertebral column. The injury may fracture the face, the vault, the base or the temporal bone or it may not produce any bone fracture. In either case the hearing function may be impaired. A loss of hearing is encountered in many cases in which no fracture of any kind can be demonstrated. On the other hand, extensive fractures of the face or skull need not result in hearing impairment.

As I[1] pointed out in 1928, in the classical animal experiments of Stenger, Brunner and others in which rats and guinea pigs were submitted to head trauma of insufficient force to fracture the skull, hemorrhages of varying degree were produced in the internal ear. These hemorrhages were found in the region of the round window and in the basal coil of the cochlea in the slightly injured animals and when the injury was more severe the hemorrhages involved both endolymph and perilymph, extending sometimes almost to the helicotrema. In these animals the round window was torn in some cases and hemorrhages were even found in the ampullae and between the fibers of the acoustic nerve. The effects of such a head injury which does not fracture the skull or the temporal bone may well be designated as a concussion of the internal ear or commotio auris.

By analogy and also from autopsy reports of Theodore,[2] Manasse,[3] Alexander,[4] Ulrich,[5] Sakai[6] and Lange[7] we can say that a similar pathologic change takes place in human ears. We may wonder what happens to the blood which has escaped into the perilymph and endolymph of the cochlea. It may and frequently does absorb, leaving no damage to the cochlear function. On the other hand, it may organize with the formation of fibrous or fibro-osseous tissue, resulting in gradual atrophy and degeneration of the neuro-

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epithelium. In the first instance the individual recovers from a slight or serious injury to his head with no loss of hearing. In the second instance there may be a hearing impairment of varying degree which may remain more or less stationary or which may become progressive, in the sense of Manasse's use of the term. Therefore, we cannot foretell in any given case whether a hearing loss will ensue, whether it will be slight or marked, and whether it will remain stationary or become progressive.

My problem is the study of traumatic impairment of hearing resulting from force applied to the head, exclusive of gunshot wounds. For the purpose of this study I must first define what I mean by a head injury. Almost everyone sustains at some time during his life a bump to the head which leaves no residual sequelae. Within the meaning of this presentation I will define a head injury as one which produces certain definite symptoms with demonstrable signs of more than evanescent character. Among such signs are fractures of the head or facial bones, unequal pupils, diplopia, strabismus, loss of visual fields, facial paralysis, spontaneous or induced signs of vestibular disturbance, fracture of the external canal wall, the escape of blood or cerebrospinal fluid from the ear, nasal hemorrhage, discoloration or suggillation about the eyes or a slowly developing hematoma over the mastoid process when this region has not been directly traumatized. I have culled 832 cases which meet these requirements from some 4000 head injuries which I have had under my observation. These 832 cases include 15 cases of transverse fracture of the petrous pyramid, 25 cases of bilateral longitudinal fracture of the temporal bone, and 92 cases of unilateral longitudinal fracture of the temporal bone. In the remainder no fracture could be demonstrated.

It must be remembered that almost all head injuries have medicolegal implications and that any claim for disability must be checked and double checked. The otologist who examines these patients must be a good psychologist and well versed in every known test for malingering. It has been contended by some that any traumatic hearing impairment due to a head injury must manifest itself shortly after the patient recovers from shock and in general I believe that this contention is correct. However, I have found that certain young individuals initially complain only of tinnitus, a tinnitus which in subsequent weeks and months associates itself with a gradually developing deafness. With such a history the hearing impairment must, I think, be ascribed to the injury, especially if it occurs in young individuals.

As the hearing function has an extensive and bilateral representation in the brain, it is inconceivable to regard the hearing defects caused by a head injury as due to pathologic conditions in the hearing centers themselves. The condition causing the hearing loss is, therefore, always in the end organ. A transverse fracture of the petrous pyramid causes a total destruction of hearing on the side of the lesion. The opposite ear may escape without any hearing loss or may develop a perceptive type of deafness. In some of the longitudinal fractures of the temporal bone the hearing impairment is conductive in type because of derangement of the middle ear structures. Even in these cases, as Skoog[9] has pointed out, the hearing defect may later become perceptive in type. In all other craniocerebral injuries in which no bone fracture is found the hearing defect is either a pure perception deafness or a mixed type.

In my whole series of 832 cases I found 228 individuals with traumatically damaged hearing, or 27.5 per cent. We can, therefore, say that better than one out of every four persons who sustain a real head injury in the sense of this presentation will develop loss of hearing. This total of 228 patients included 15 with total deafness of the affected ear, 185 with a pure perceptive or with a mixed type of impairment, 16 with a pure conduction deafness and 12 cases in which an aggravation of a pre-existing deafness occurred.

The age factor is of considerable importance in the development of hearing impairment after head injuries. In the internal ear concussion group where no fracture could be demonstrated there were 13 with a pure perceptive type of impairment under 30 years of age, 43 between the ages of 30 and 45 years, and 52 in individuals over 45 years of age. Among these internal ear concussion cases there were two individuals with a pure conduction deafness and 22 with a mixed form of deafness. The two with conduction deafness are questionable because the middle ear was not involved in any known traumatic process.

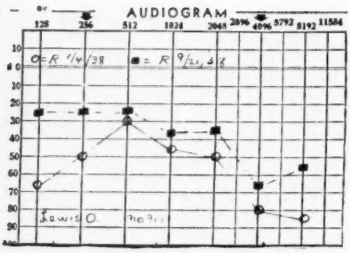
In the group with longitudinal fractures of the temporal bone, either unilateral or bilateral, there were 14 persons with a pure conductive type of impairment, 55 with a perceptive type of loss, and 48 with no hearing loss whatsoever. In this group the age factor was not so important, there being 21 individuals under 30 years of age, 24 between the ages of 30 and 45, and 24 who were over 45 years of age. If one now considers the age factor in both the internal ear concussion and the temporal bone fracture groups, one finds that there were 34 under 30 years of age, 67 between the ages of 30 and

45, and 76 who were over 45 years of age. I believe, therefore, that we can generalize and say that the likelihood of an individual suffering a hearing loss after a head injury is, in general, in direct proportion to his age. Considering the question as to whether the hearing loss will be unilateral or bilateral I found that the patients with traumatic hearing impairment were about equally divided. One would naturally suppose that when the injury was severe enough to fracture the temporal bone the likelihood of a hearing defect would be greater than when no such fracture occurred but this is not necessarily true. In 10 out of 25 cases in which both temporal bones sustained a longitudinal fracture, as evidenced by bleeding from both ears, no hearing loss was found. This was also true in 34 out of 92 cases of unilateral fracture of the temporal bone.

In the internal ear concussion group there were six individuals who sustained such a marked hearing impairment in one ear that I designated the loss as subtotal. In these cases there was no damage to the hearing of the opposite ear. I have defined a subtotal loss as one with an average loss of more than 80 decibels for the frequencies of 512, 1024, and 2048. Likewise, in the group of unilateral longitudinal fracture cases there were six such cases. The lesion in these 12 cases must have consisted of a massive hemorrhage into the cochlea or damage to the cochlear nerve itself.

The question of the aggravation of a preexisting deafness by a craniocerebral injury is a very interesting one. If a head injury, with or without fracture, can damage the normal hearing, there is no logical reason why it cannot aggravate the hearing impairment already existing at the time of the injury. However, we must approach this question with a very critical mind, remembering always that most head injury cases are medicolegal in character and that the claim of aggravation of an already existing hearing loss is hard to prove or disprove. This is especially true in those individuals of whom we have no accurate records of the preexisting deafness. Bearing all this in mind I have selected 12 cases from my records in which I felt morally certain that a preexisting hearing defect had been aggravated by the head injury. In six cases the aggravation was unilateral and in six bilateral. As is to be expected, this aggravation occurred in older individuals. With the exception of one boy of 22 these people were all over 45 years of age.

What is the course of the hearing lesion produced by craniocerebral trauma? It has been observed by numerous authors (Alexander and Scholl,[8] Skoog,[9] Ulrich[5] and Permin[10]) that an early



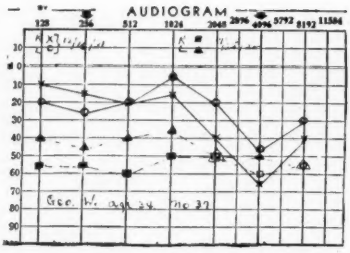


Fig. 1.—Audiograms of the right ear of Lewis O., aged 29, made Jan. 4, 1938, and Sept. 21, 1938. Left ear normal. No fracture.

Fig. 2.—Audiograms of George W., aged 34, made Dec. 16, 1931, and July 25, 1932. Bilateral perceptive deafness. No fracture found.

hearing loss which occurs after a traumatic skull injury tends for a time to improve, in some cases even to normal; then after a variable length of time a more gradual deterioration sets in. Skoog[9] examined 69 patients with longitudinal fractures of the temporal bone in whom the immediate findings were as follows:

Affection of the sound conduction apparatus	65%
Affection of the sound perception apparatus	22%
A mixed form of deafness	13%

He reexamined the hearing of these individuals seven to thirteen years later and then found:

Normal hearing in	30%
Conduction deafness in	16%
Perception or mixed deafness in	54%

The explanation for this seems to be that the interference with sound conduction clears up when the pathologic condition in the middle ear improves. On the other hand an injury which has produced hemorrhage into the cochlear spaces or damaged the auditory nerve is not so apt to improve but may result in further deterioration of the hearing. In an attempt to find out what does happen in a traumatically produced hearing impairment, I have assembled 51 cases in which I have been able to make two or more examinations at shorter or longer intervals. Of these 51 cases the hearing im-

proved in 20 (Fig. 1). In 23 cases the hearing deteriorated further (Fig. 2) and in eight cases the hearing remained stationary. Eleven of these 51 cases were longitudinal fractures of the temporal bone. Of these, in five the hearing improved on subsequent examinations, in five the hearing became worse, and in one it remained stationary. The ratio between those cases in which the hearing will deteriorate and those in which it will improve or remain stationary is about the same in the temporal bone fracture cases as it is in the internal ear cases. Combining both groups of cases we can say that there is about as much chance of the hearing improving as there is of further deterioration.

In summary, therefore, we can say that of those cases of craniocerebral trauma with demonstrable signs of injury better than 25 per cent will develop hearing impairment. Of these, 81 per cent will exhibit either a pure perceptive type of hearing loss or a mixed form. We can also say that the probability of a traumatic hearing loss increases with age. We cannot say whether the traumatically produced deafness will be unilateral or bilateral. A hearing impairment of such marked degree that it can be called subtotal can occur without any demonstrable evidence of bone fracture and is, I believe, due to hemorrhage into the cochlear spaces with subsequent organization. My study has demonstrated that an aggravation of a preexisting deafness can take place in older individuals. My study has also demonstrated that a traumatically produced hearing defect has just as much chance of remaining stationary or improving as it has of further deterioration.

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XXIX

CHORDOMAS OF THE CERVICAL REGION

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These rare tumors that are vestigial remnants of the notochord[1] (an axial structure characteristic of all vertebrates) are usually located at those areas representing its extremities, viz., the spheno-occipital synchondrosis and the sacrococcygeal region. Rarely are they found in the cervical areas.

Normally portions of the notochord persist in the intervertebral discs as the central mucoid cores or pulpy nuclei (nuclei pulposi).[2]

Virchow[3] in 1857 described small jelly-like tumors occurring in the region of the dorsum sellae and named them "ecchondrosis physaliphora." These were attributed to islets of degenerated cartilage from the spheno-occipital synchondrosis.

Fabricius[4] reported a case of a patient with a chordomatous growth of the nasopharynx and pharynx, who was operated upon twice (the second operation being four and one-half years after the first) and who remained well without further recurrence. Complete details are not given.

Syme and Cappell[5] described in some detail such a tumorous growth. It was located on the posterior pharyngeal wall and grew upward toward the nasopharynx and downward toward the larynx, being attached to the second, third and fourth cervical vertebrae. The chief symptoms were pain in the head and neck, stiffness in the neck and difficulty in swallowing. The patient was operated upon in July 1926, and by January 1927 a second operation became necessary but was unsuccessful. An external approach was used. There was no lymph node involvement and no invasion of the adjacent structures was observed at the operation done in January. However, one lymph node was found to be involved at the second operation.

Andre-Thomas and Villandre[6] reported a case, and Cappell[7] reported two such tumors attached to the cervical vertebra. Chiari[8]

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reported a cervical chordoma which was attached to the surfaces of the borders of the fourth and sixth cervical vertebrae.

Definitely, chordomas of the pharyngeal and midcervical areas are of such uncommon occurrence that they warrant recording, and such a report will be included in this communication.

Ridpath[9] states these tumors "have been reported as occurring in various locations in the following order of frequency: in the sacro-coccygeal in ninety-one cases; in the spheno-occipital region in fifty-two cases; in the cervical portion of the spine in twelve cases, and in other locations, rarely."

Livingstone[10] groups these tumors into three classifications.

"*Type* 1. The small tumors called by Virchow 'ecchondrosis physaliphora.' They occur in two percent of postmortem examinations and are composed of the same type of cells as the nucleus pulposus of the intervertebral discs.

"*Type* 2. The malignant tumor in which active mucin-producing cells predominate and in which recurrences and metastases occur.

"*Type* 3. The main feature of this type is the production of cells which resemble cartilage and the tumors are usually encapsulated, and do not recur."

A great many of the tumors reported as occurring in the cervical area have been high, usually arising from the first and second cervical vertebrae, and they were usually associated with growths in the spheno-occipital synchondrosis. Very complete and detailed articles by Owne, Hershey and Gurdjian,[11] by Haas[12] and by Adson, Kernchan and Waltman[13] have dealt mostly with cranial and high cervical tumors.

Many authors have expressed varying opinions as to the cause and origin of these tumors. It would seem that there is unanimity of opinion that they originate from displaced notochordal remnants. The cause of their growth has not been satisfactorily explained, for Stout[14] reports a maxillary chordoma in a day-old infant and the patient on whom this report is being made is 63 years old.

The exact diagnosis cannot be made without biopsy. However, a slow-growing tumorous mass appearing in the oropharynx or the laryngopharynx, producing pressure symptoms on the adjacent nerves of the cervical plexus, disturbance in breathing and deglutition, caus-

ing no fever, and on palpation having a tense jelly-like feel, should be suspected as possibly being a chordoma of this region.

X-ray studies will show a bulging shadow of varying density in the pharynx. With barium, the shadow is very clearly outlined. Also, any involvement of the bodies of the vertebrae and the intervertebral discs may be determined. The amount of obstruction in the food and air passages may also be evaluated.

Aspiration biopsy will, very likely, give negative results, excepting that if the tumor mass contains matter that is more fluid than semisolid a diagnosis may be arrived at.

Cysts and retropharyngeal abscess must be differentiated.

Stout[15] epitomized the microscopic findings by stating that chordomas are characterized by the so-called physaliphorous cells which are polyhedral, with a small nucleus, cytoplasm much distended with glycogen and vacuoles containing a mucin-like material. This gives to the tumor its characteristic jelly-like appearance.

The desiderata necessary to diagnose a chordoma has been postulated by Linck[16]:

1. Syncytial giant cells.
2. Curious concentric nests of cells.
3. Nuclear vacuolation, the cell possibly filled with glycogen.
4. Physaliphorous cells (the nucleus is located peripherally and the cytoplasm is filled with mucinous globules).
5. Irregular trabeculae of tumor cells separated by abundant intercellular mucinous matrix.

It has been refuted by many who have studied these tumors that the substance within the much distended cell is glycogen, the chemical test having been confused by the fixative.

This tumor is invasive and very resistant to roentgenotherapy. Its anatomical situation is such that the external surgical approach is difficult.

Suspension laryngoscopy gives an excellent exposure of the pharynx and the laryngopharynx and should be the method of choice in the excision of such a tumor, located between the third and sixth cervical vertebrae.

The mucous membrane, submucosa and pharyngeal aponeurosis may be dissected from the tumor and, excepting where it is attached

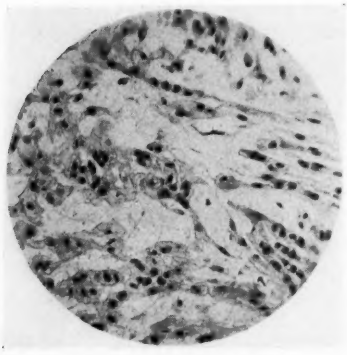


Fig. 1.—Photomicrograph of the chordoma (4 mm. obj: 6 x occ.) showing the cells arranged in strands and many physaliphorous cells—-large cells with distended cytoplasm and small nuclei.

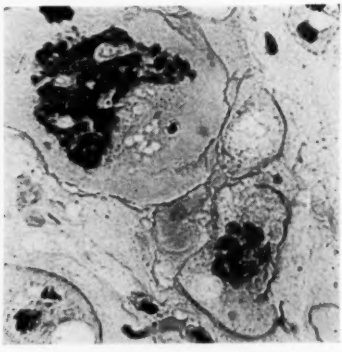


Fig. 2.—Photomicrograph (1 8 mm. obj: 6 x occ.) showing physaliphorous cells with distention of the cytoplasm, vacuolation and pyknosis.

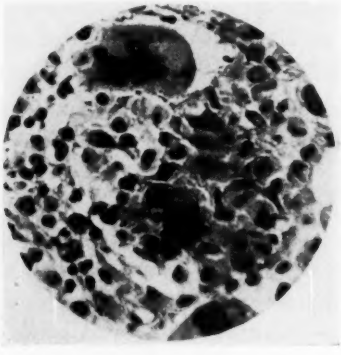


Fig. 3.—Photomicrograph (4 mm. obj: 6 x occ.) showing giant cells.

to the bodies of the vertebrae, may be removed intact. These tumors are usually well encapsulated.

The following case report is briefly detailed:

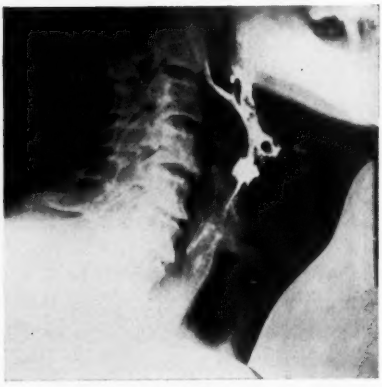
REPORT OF A CASE

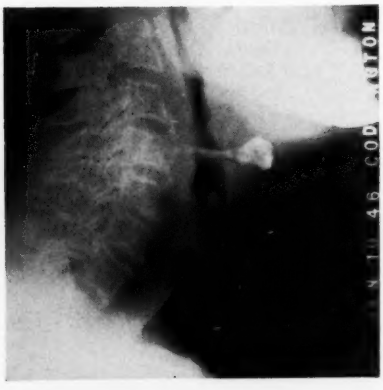
The patient, W. C., male, aged 62, American, stated his father died when 76 years old from a tumorous growth of the rectum that necessitated a colostomy. Up to June 1945, the patient had had no serious illness. About that time, he noticed that he was having slight difficulty in swallowing but attributed this to the trouble he was having with his teeth. Recently, the difficulty in swallowing had increased so that he experienced a sensation of complete obstruction, particularly when the larynx was drawn up by the hyoid muscles during the act of swallowing. He stated that it felt as though a "ball valve" closed.

During the past few months, he had lost considerable weight (40 lbs.) and his strength and vigor had diminished accordingly. There had been no pain or pressure symptoms on any of the adjacent nerves.

The general appearance of the patient was that of an emaciated elderly male, very apprehensive, and somewhat uncooperative. All of his teeth were in a state of partial or total decay, the gums were swollen and inflamed, and there were many areas of superficial ulceration of the mucous membrane of the mouth. The epipharynx and the nostrils were negative.

By indirect laryngoscopy, there was seen an obstructing mass slightly to the right of the midline. It was smooth in outline, covered by the normal mucous membrane of the pharynx and without irregularities or ulcerations. The growth was approximately at the upper level of the epiglottis and extended downward on the posterior wall. The larynx, including the epiglottis, was seen by indirect laryngoscopy when the chin was elevated and the neck extended. The vocal cords were visualized in their entirety to the anterior commissure. The ventricular openings appeared to be normal. External palpation showed the larynx movable but not freely so, and no crepitations were felt on the spinal column. The mass within the hypopharynx could not be palpated externally with any degree of certainty as to its outline. There were no enlarged lymph nodes. Digital palpation revealed that the hypopharynx was almost completely obstructed.





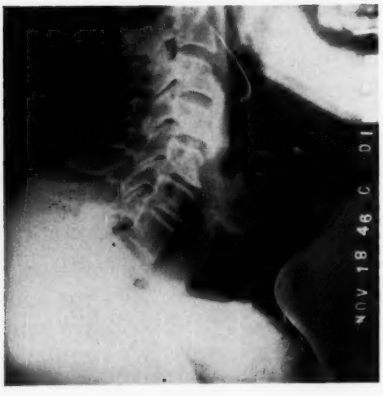


Fig. 4.—Lateral roentgenogram taken November 7, 1945 showing a shadow extending forward from the vertebral region. The upper margin of the bulge is at the top of the third cervical body. The apex of it is level with the middle of the fourth. The normal thickness of the tissue resumes at the level of the sixth. Barium mixture outlines the tumor mass.

Fig. 5.—Roentgenogram taken January 10, 1946. On November 7, 1945, the roentgenogram showed a very prominent bulging forward of the posterior pharyngeal wall centering over the fourth cervical body. There is no bulge as of this date.

Fig. 6.—Roentgenogram taken November 18, 1946. On November 7, 1945, there was a soft mass the apex of which was level with the middle of the fourth cervical body and the upper margin at the level of the top of the third. There is nothing now (November 18, 1946) that suggests any recurrence of the tumor which was removed.

There is a shadow of bony density and texture that is apparently calcification originating in the third and fourth cervical bodies and which seems to bridge the ventral surfaces of these two. At the first examination I noted a rarefaction of the ventral surface of the fourth body, and it seems reasonable that this calcification may be the end stage of some process that was going on in it at that time. The retropharyngeal wall measures on the film only 3 mm. in thickness over this calcification. (R. Spillman, M.D., roentgenologist.)

The temperature range was from 98° to 99° F.

The tentative diagnosis was a soft tumor of the hypopharynx, possibly a cyst. We advised roentgenograms, aspiration biopsy and biopsy.

The x-ray films showed the extent of the tumor to be from the third to the sixth cervical vertebra and that it protruded forward. The meager report was helpful in that it showed the extent of the obstructing mass but nothing else except in a negative way—that there was no osteomyelitis of the spine or tuberculosis.

Aspiration of the tumor was productive of no data.

Under local anesthesia, a specimen was taken from the center of the tumor for biopsy. The opening into the pharyngeal tissue was less than 5 mm. There was no reaction following this procedure. The patient was given sulfadiazine, and after two days was permitted to leave the hospital in order to adjust his affairs, have his decayed teeth removed and return to the hospital within two weeks.

The pathological report of the biopsy was somewhat confused but, after consultation with several pathologists, it was decided that it was a chordoma and that this would be confirmed by the gross and microscopic examinations of the tumor when it was removed.

On November 19, 1945, at the Manhattan Eye, Ear & Throat Hospital, the tumor was removed with the patient in the suspension laryngoscope. Ether anesthesia was used. The procedure consisted of making a transverse incision of the mucous membrane and the subadjacent tissues over the greatest prominence of the tumor and separating a rather thin-walled capsule of the tumor from the adjacent structures. The tumor was attached to the region of the fourth cervical vertebra and it was necessary to use sharp dissection in order to free it. The tumor mass was delivered from its bed almost intact. The posterior attachment was somewhat ragged. There was comparatively little bleeding and this was controlled by pressure. There were no distinct bleeding points, the bleeding being of the capillary type.

A nasal feeding tube was inserted and the patient returned in very good condition. Within 18 hours the patient was out of bed for a short period (5 minutes) and within five days the feeding tube was removed. He was able to leave the hospital November 25, 1945.

For four days after operation, he received 25,000 units of penicillin every 3 hours.

The pathological report was as follows:

Gross: A gelatinous mass is enclosed in a fibrous capsule approximately 5 x 4 x 2 cm. with fibrous bands running through it and attaching themselves to the capsule. It has a slightly reddish color and a peculiar odor.

Microscopic: Sections show that there is a tumor made up of peculiarly large, polygonal cells which are arranged sometimes in cords and are surrounded by mucoid material. The individual cells sometimes attain a gigantic size and some are vacuolated, producing a physaliphorous effect. The tumor grows diffusely and tends to infiltrate. This seems to be a characteristic chordoma in one of the less common situations.

Diagnosis: Chordoma of the hypopharynx. (Eggston, Stout)

SUMMARY

A chordoma situated in the cervical region (third to sixth vertebra) is described, together with the diagnostic procedure and the method of treatment.

There has been no recurrence to date, and the patient is in good physical condition.

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XXX

A STATISTICAL STUDY OF POLIOMYELITIS IN RELATIONSHIP TO TONSILLECTOMY

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During the past 20 years and particularly the past decade, poliomyelitis has assumed a position of particular importance. Although first described by Jacob Von Heine in 1840, it was not until the twentieth century that universal scientific efforts were directed to the study of this disease.

During the Springfield, Massachusetts, epidemic of 1910, Sheppard[12] reviewed 200 cases of the disease and discovered that one case of bulbar type poliomyelitis had followed a recent tonsillectomy. He also noted that, "Since the exact function of the tonsil is still a matter of speculation, I have sought to collect some data as to the possible significance of its presence or absence in cases of acute epidemic poliomyelitis." No doubt the quotation might well be repeated today with very little exception. From 1910 until 1946 no less than 314 cases of poliomyelitis following tonsillectomy have been reported in the literature (Table 1). This does not include 11 cases covered in this study. The reports available indicate that physicians in Great Britain,[41] Australia,[10] and Argentina[21] have recorded cases of poliomyelitis following tonsillectomy. This is quite understandable when we realize that we are dealing with a widespread but generally limited disease.

Sabin[11] experimentally reproduced poliomyelitis in the Rhesus monkey by tonsillopharyngeal injection of 100-1000 minimal cerebral infective doses. Other attempts to reproduce the disease by swabbing the virus suspension on recently tonsillectomized areas in the monkey were not successful. However, the Cynomolgus monkey, as used in later experiments by Sabin,[44] Faber and Silverberg,[31] and others, more nearly resembles the human as regards infectibility, and nontraumatic methods of infection via oronasal spray and inhalation

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readily produce poliomyelitis. Still later experiments by Faber, Silverberg and Dong[38] reproduced poliomyelitis in both Rhesus and Cynomolgus type monkeys by inhalation of suspensions of dry droplet nuclei through a special inhalation chamber.

Howe, Bodian and Wenner[20] succeeded in transmitting poliomyelitis directly from patients to Rhesus monkeys. The virus was recovered from the oropharynx of poliomyelitis patients by use of sterile cotton swabs and later injected into Rhesus monkeys via the intracerebral route. In 36 cases of poliomyelitis the virus was recovered in 10 cases, or 28 per cent. Again it must be borne in mind that the virulence of many strains of virus varies greatly and experimental workers[40] have recovered strains with extreme virulence in contrast to earlier experimental varieties.

Faber, Silverberg and Dong[38] have concluded that the nasal mucosa was highly vulnerable in the Cynomolgus monkey to poliomyelitis virus. Faber et al concluded that the infection is nerve-borne from the mucous surfaces to the appropriate peripheral ganglia and thence to the central nervous system. It was further stated that the presence of bare nerve endings was not essential to the penetration of the virus.

In the examination of the peripheral and central nervous tissues of eight patients dying of acute poliomyelitis, Faber and Silverberg[34] concluded that the pharynx appeared to be an especially favorable site for the primary penetration of the virus into the body. In this neuropathological study the trigeminal (5th cranial) and visceral afferent (9th and 10th cranial) systems were predominately involved. In contrast to earlier experimental evidence with monkeys it was noted that no involvement of the olfactory bulb was found. However, it was thought that the virus failed to reach the olfactory mucosa in infective amounts at the time infection is initiated.

Theoretical factors governing the possible invasion of the neural tissue have been set forth[32, 16] on the basis of reduced resistance following anesthesia, nutritional deficiency and anoxia. The specific affinity for neural tissue on the part of the virus together with the known effect[32, 42] of anoxia on the nervous system warrants consideration of these theories.

The virus of poliomyelitis is believed to be widely disseminated throughout the general population during periods of epidemics and is commonly recovered from stools of asymptomatic carriers during these periods. However, Kessel and Moore[28] during an interepidemic

period recovered poliomyelitis virus from both tonsils and stools in five of 136 people tested during a period from May to September 1942. Late experimental work[38, 39] would tend to favor the upper alimentary tract (pharynx) as a portal of entry, while earlier reports[31, 43, 44] favor the lower alimentary tract as a site of entry.

Following the now famous "K" family in Akron, Ohio,[9, 30] nationwide concern became apparent in the unusual space devoted by daily newspapers and magazines to this tragic occurrence. The disease was viewed by the public at large with considerable apprehension, due, no doubt, to its rapid epidemic tendencies, the grave paralytic sequelae and the inability of the medical world to bring forth a suitable and comforting panacea. As is so often the case, a great deal of generalizing resulted from this impressive demonstration of what can occur when poliomyelitis follows a tonsillectomy. Notable of these was the deferment of tonsil surgery during the epidemic periods. Although no other solution appears imminent, the interepidemic phases have lessened the risk and only occasional episodes arise to alert us to the potential hazards involved.

A recent paper on the topic of poliomyelitis and tonsillectomy[32] advocated a nationwide survey on the part of otolaryngologists to determine the incidence of poliomyelitis following tonsillectomy.

This report began as a part of that survey and expanded in scope as the material became available. This study was limited to the files of the San Francisco City and County Hospital* and the Children's Hospital** of the same city. All facilities for hospitalization of poliomyelitis cases in the city of San Francisco are centered in the contagion wards of these two hospitals. It was thought that a representative period for study should include an epidemic and an interepidemic period, if possible, with no break in yearly sequence. The period from 1941 to 1945, inclusive, seemed appropriate and was therefore selected for study. The material gathered from the files of the two hospitals covered the following points:

1. Age.
2. Address.
3. Date of onset of poliomyelitis.
4. Type of poliomyelitis: bulbar, bulbospinal, spinal, or nonparalytic.

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*Operated by the joint staffs of the University of California and Stanford University Schools of Medicine.

**Information was obtained through the courtesy of Drs. E. B. Shaw and A. Palermo.

5. Any operation performed during a two-month period before onset of disease.
6. Outcome: recovery, paralysis, or death.

In those instances where doubt existed as to the authenticity of the data sought, the case was omitted from this study.

In a further effort to correlate the material gathered herein, an attempt was made to ascertain the number of tonsillectomies performed during the same period. A form letter was directed to hospitals in 34 counties in which the cases of poliomyelitis had originated that were hospitalized in one or the other of the hospitals surveyed. Out of 96 letters mailed out, 63 replies were received. No attempt was made to contact physicians to determine the number of cases operated on in their own offices. From the replies received, the known number of tonsillectomies and adenoidectomies performed for the five-year period was tabulated as follows:

1941: 11,301

1942: 12,406

1943: 8,910

1944: 12,889

1945: 12,290

A total of 57,796 known operations were thus performed during the five-year period from 1941 to 1945, inclusive.

For the same 34 counties the following cases of poliomyelitis were reported to the California State Department of Public Health:

1941: 58

1942: 52

1943: 1,275

1944: 177

1945: 495

Thus, for the five-year period 2,057 cases of poliomyelitis were reported for the area surveyed. During the five-year period, from 1941 to 1945, 492 cases of poliomyelitis were hospitalized in the two hospitals covered in this study (Table 2). The epidemic years of 1943 and 1945 accounted for the majority of cases. The population

for the 34 counties covered in this study was listed as approximately 2,500,000 by the United States Census of 1940. Thus, during the greatest epidemic year, 1943, the incidence of poliomyelitis to the general population was approximately 1 to 1960.

The age incidence of poliomyelitis in 492 cases covered in this series (Table 4) indicates that the majority of cases occurred between the ages from 2 to 9. The greatest number, which was 40, fell at age 8. The seasonal distribution of poliomyelitis in 492 cases (Table 5) indicated that the majority of cases fall into a seven-month period, from June to December. June produced 36 cases; July, 73; August, 79; September, 80; October, 81; November, 71 and the number in December fell to 24. Of the 492 cases surveyed, 354 cases were recorded as to known presence or absence of tonsils before onset of the disease. The cases (Table 3) were catalogued by clinical type, also indicating the ultimate outcome in the series as to paralysis, recovery or death. Undoubtedly, the number of paralytics was reduced as time went on, but the figures taken represent the available information at the time of discharge. In the group with tonsils present there were 6 deaths, while in the group with tonsils absent there were 11 deaths. There were 11 cases of bulbar and bulbospinal type of disease in the group with tonsils present. There were 24 cases of bulbar and bulbospinal type poliomyelitis in the group with tonsils removed.

Of the total of 492 cases reviewed in this series, there were a total of 11 (Table 6) cases of poliomyelitis following tonsillectomy within a two-month period. The longest interval between operation and onset of poliomyelitis was 60 days, the shortest being 7 days. There were 6 cases of bulbar and bulbospinal type of poliomyelitis following tonsillectomy in this series. There were 4 cases of the spinal type and one case was classified as unknown by type. There were 7 males and 4 females in this series. The oldest patient was nine years of age and the youngest three years of age. There were no deaths in this series of cases of poliomyelitis following tonsillectomy within 60 days.

SUMMARY

1. A review of 492 cases of poliomyelitis hospitalized in San Francisco during 1941 to 1945 is presented.

2. The cases reviewed were found to have originated from 34 counties in the State of California, which reported 2,057 cases of poliomyelitis during the five-year period for the same counties.

3. A survey of hospitals in the 34 counties concerned reveals a total of 57,796 known tonsillectomies performed during the five-year period.

4. In one group of patients with tonsils present, there were 6 deaths, while in another group with tonsils removed there were 11 deaths.

5. In the group with tonsils present there were 11 cases of bulbar and bulbospinal type poliomyelitis, while in the group with tonsils removed there were 24 cases of bulbar and bulbospinal poliomyelitis.

6. Of 492 cases of poliomyelitis surveyed, there were 11 cases of poliomyelitis following recent tonsillectomy. There are 314 cases of poliomyelitis following tonsillectomy reported in literature heretofore.

7. Of 11 cases of poliomyelitis following tonsillectomy, 7 cases occurred within the probable period of incubation.

8. Neuropathological studies at autopsy in 8 cases strongly suggest a pharyngeal portal of entry.

CONCLUSIONS

From our study of these cases we find.

1. That the incidence of poliomyelitis to general population in an epidemic year (1943) was (ratio) 1 to 1,960, while the incidence of poliomyelitis following tonsillectomy for the same period was (ratio) 1 to 1,782 (5 cases in 8,910 known tonsillectomies).

2. That the incidence of poliomyelitis following recent tonsillectomy is not greatly out of proportion to the ratio of disease to the general population during an epidemic year.

3. That when poliomyelitis occurs following tonsillectomy it is more apt to be bulbar in type.

4. That there is a higher incidence of bulbar and bulbospinal type poliomyelitis in tonsillectomized patients than in nontonsillectomized patients, the ratio being two to one.

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TABLE 1.

POLIOMYELITIS CASES FOLLOWING TONSILLECTOMY REPORTED TO DATE (1946).

WITHIN 30 DAYS					30 TO 60 DAYS						
B	BS	UNK	S	NP	B	BS	UNK	S	NP	TOTAL	REFERENCES
1	—	—	—	—	—	—	—	—	—	1	Sheppard 1910[12]
—	—	—	—	1	—	—	—	—	—	1	Boyd 1912
9	—	—	—	—	—	—	—	—	—	9	Ayer 1928[1]
7	5	—	4	—	—	—	—	—	—	16	Aycock and Luther 1929[2]
4	—	2	—	—	—	—	—	—	—	6	Gordon 1931
5	—	—	—	—	—	—	—	—	—	5	Silverman 1931[3]
4	—	—	2	3	—	—	—	—	—	9	Ontario Health Dept. 1937
2	—	—	—	—	—	—	—	—	—	2	Anderson 1938[41]
15	2	—	4	3	1	—	—	3	1	29	Eley-Flake 1938[4]
6	3	—	2	2	—	—	—	—	—	13	Stillerman Fischer 1938[5]
1	—	—	—	—	—	—	—	—	—	1	Pamment 1933[8]
1	—	—	—	2	—	—	—	—	—	3	Kramer Gilliams 1938
1	—	—	—	—	—	—	—	—	—	1	Stebbins-Gillick Ingraham 1939
2	—	—	—	—	—	—	—	—	—	2	Koskoff 1939[7]
5	2	—	2	3	2	—	—	13	—	27	Fischer-Marks-Stillerman 1941[6]
2	3	—	1	1	—	—	—	—	—	7	Helms 1941[10]
5	—	—	—	—	—	—	—	—	—	5	Krill-Toomey 1941[9]
15	5	5	5	3	5	—	4	11	—	53	Vt., 1912-31 Mass., 1927
14	2	—	2	2	—	1	1	3	—	25	Personal Communications
3	—	—	3	—	—	—	—	—	—	6	Howard[29]
17	—	—	—	—	—	—	—	—	—	17	Anderson 1945[14]
13	—	—	3	1	—	—	—	—	—	17	Krill-Toomey 1942[15]
4	—	—	2	—	3	—	—	3	—	12	Kansas 1940-43[29]
3	—	—	1	—	—	—	—	—	—	4	Wynn's 1943[29]
8	2	—	2	—	1	1	—	—	1	15	Pisczek 1943[29]
—	1	—	4	—	—	—	—	—	2	7	McFarland 1943[29]
—	—	6	—	—	—	—	2	—	—	8	Vasquez 1944[21]
—	—	4	—	—	—	—	—	—	—	4	Cunning 1946[32]
3	—	—	—	—	—	—	—	—	—	3	Lubchenko 1943[35]
—	—	6	—	—	—	—	—	—	—	6	Roberts 1946[33]
150	25	23	37	21	12	2	7	33	4	314	

B—Bulbar
BS—Bulbo-spinal
S—Spinal
UNK—Unknown
NP—Non-paralytic

Adapted from
Aycock[26]
Howard[29]

TABLE 2.

POLIOMYELITIS CASES SURVEYED FROM THE FILES OF CHILDREN'S HOSPITAL AND SAN FRANCISCO CITY AND COUNTY HOSPITAL 1941-1945 INCLUSIVE

SAN FRANCISCO CITY AND COUNTY HOSPITAL

TYPE	1941	1942	1943	1944	1945	TOTAL
Bulbar	..	....	5	....	2	7
Bulbo-spinal	..	....	4	....	5	9
Spinal	1	3	71	13	38	126
Non-paralytic	..	1	7	13	16	37
Total:	1	4	87	26	61	179

CHILDREN'S HOSPITAL*

TYPE	1941	1942	1943	1944	1945	TOTAL
Bulbar	1	1	16	6	1	25
Bulbo-spinal	..	1	15	5	5	26
Spinal	6	8	147	18	56	235
Non-paralytic	..	....	10	3	14	27
Total:	7	10	188	32	76	313

*Data obtained through the courtesy of Dr. A. Palermo, Children's Hospital, San Francisco, California.

TABLE 3.

POLIOMYELITIS ACCORDING TO KNOWN PRESENCE OR ABSENCE OF TONSILS, THE CLINICAL TYPE OF DISEASE AND ULTIMATE OUTCOME 1941-1945 INCLUSIVE**

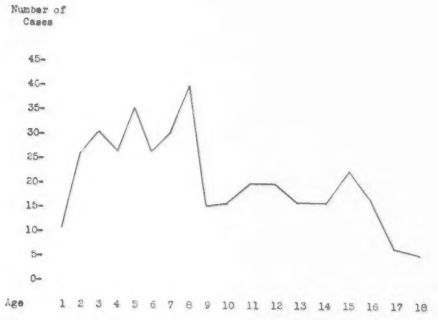
	TONSILS PRESENT					TONSILS ABSENT				
TYPE*	B	BS	S	NP	TOTAL	B	BS	S	NP	TOTAL
Deaths	3	1	2	....	6	8	....	3	....	11
Paralysis	2	3	96	..	101	4	12	96	....	112
Recovery	..	2	66	31	99	..	..	..	25	25
Total	5	6	164	31	206	12	12	99	25	148

*B—Bulbar
S—Spinal
BS—Bulbo-spinal
NP—Non-paralytic

**Hospital records Children's Hospital and San Francisco City and County Hospital, San Francisco.

TABLE 4.

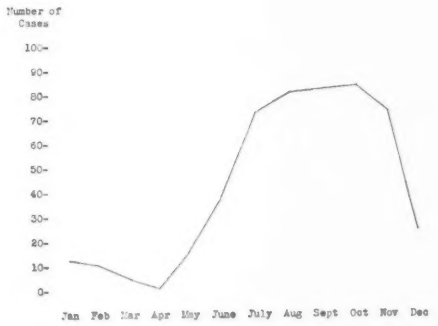
AGE INCIDENCE FROM ONSET IN 492 CASES OF POLIOMYELITIS DURING 1941-1945 INCLUSIVE (SAN FRANCISCO).



Combined Statistics From Children's Hospital and San Francisco City and County Hospital.

TABLE. 5.

SEASONAL DISTRIBUTION FROM DATE OF ONSET IN 492 CASES OF POLIOMYELITIS DURING 1941-1945 INCLUSIVE (SAN FRANCISCO).



Combined Statistics From Children's Hospital and San Francisco City and County Hospital.

TABLE 6.

DATA ON CHILDREN IN WHOM POLIOMYELITIS DEVELOPED WITHIN 60 DAYS AFTER REMOVAL OF TONSILS AND ADENOIDS***

	PATIENT	AGE**	DATE OF OPERATION	DATE OF POLIO ONSET	INTERVAL DAYS	TYPE OF* POLIO	OUTCOME
1.	T.D.	3f	7-13-43	7-27-43	14	B	Recovery
2.	D.L.	9m	6-11-43	7-25-43	44	UNK	Recovery
3.	G.K.	8m	6-24-43	7-24-43	30	BS	Paralysis
4.	J.A.	3f	8-6-43	8-20-43	14	S	Paralysis
5.	G.B.	7m	6-3-43	7-3-43	30	S	Paralysis
6.	C.W.	4f	6-16-44	6-23-44	7	B	Recovery
7.	C.R.	6f	9-10-44	9-24-44	14	BS	Paralysis
8.	J.H.	5m	7-21-45	8-3-45	13	BS	Paralysis
9.	A.Z.	5m	7-22-45	9-21-45	60	S	Paralysis
10.	J.L.	4m	11-7-45	11-21-45	14	S	Paralysis
11.	E.K.	8m	10-22-45	11-3-45	12	BS	Paralysis

*B—Bulbar
BS—Bulbo-spinal
S—Spinal
UNK—Unknown

**f—female
m—male

***From the files of the Children's Hospital and San Francisco City and County Hospital 1941-1945 inclusive.

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XXXI

THE SURGICAL TREATMENT OF NASOPHARYNGEAL FIBROMA

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Fibroma of the nasopharynx is a relatively uncommon tumor which occurs predominately in adolescent males. It has a definite tendency to regress in adult life. It is not malignant histologically but may become malignant clinically because of pressure or location.

Nasopharyngeal fibromata appear to arise most commonly from the periosteum covering the base and the anterior face of the sphenoid and the basilar process of the occipital bone. The tumors vary greatly in size and in extent. Grossly, the tumors represent fairly hard, firm masses of tissue which are, most frequently, firmly attached to the bony site of origin. They are very rarely pedunculated except in their late stage when they become very large. During the early stage of their development they are broadly attached to the bone of the occiput and sphenoid, and are not easy to separate. They may extend into the sphenoid sinus, the nasal fossae, the ethmoid, the antrum, the orbit, or inferiorly into the pharynx, and may extend below the soft palate and uvula. Microscopically, these tumors consist of dense collections of fibroblasts with numerous blood vessels. The vascularity is so great in some tumors that they are termed angiofibromata.

Numerous reports have appeared in the literature regarding the pathology and the treatment of this condition—notable are the writings of Allan,[1] Brunner,[2] Coates,[3] Delavan,[4] Friedberg,[5] Goldsmith,[6] Hill,[7] and New and Figi.[8, 9] When the treatment of this disease is considered, one is confronted at once by the wonderful results reported by New and Figi[8] in the use of radium and diathermy. Sixty-five cases, all successfully treated by this method, were reported in 1940. The present paper deals with the author's experience with six cases treated surgically at the Los Angeles County General Hospital and in private practice.

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There are two main surgical approaches. The first is the transnasal approach, either by Moure's lateral rhinotomy, or via the Denker route through the upper lip encircling the ala nasi. The second is the transpalatine approach which will be discussed in this paper. Jackson[10] in 1907 reported eight successful cases in which fibromata were removed through the oropharynx by forceps and snares without splitting the soft palate. He tied one or both external carotids in these cases. This transpalatine approach has been used for years but was abandoned chiefly because of postoperative infection in the suture line, resulting in perforations. This objection can now be removed by the use of penicillin. The other objection to this route was hemorrhage. Such hemorrhage can be controlled by ligature around the external carotid.

The surgical technique is as follows. Intratracheal anesthesia is preferred. The external carotid artery is exposed on the side in which there appears to be greater involvement. The ligature is placed around the vessel, but is not tied. The soft palate is incised longitudinally in the midline up to the hard palate. The incision extends inferiorly to one side of the uvula. Each palatal half is retracted laterally. The tumor mass is then freed by the fingers to the base; then a strong, flat, semisharp elevator is used to pry the growth from its attachment at the base of the skull. This is the most difficult and painstaking part of the procedure; here is where hemorrhage is most severe and where extreme care is required in separating the mass. After the base is separated, the extensions are loosened and traction is applied by a heavy snare or Brandegee forceps. If hemorrhage is not controlled by pressure, the external carotid is tied. The cavity is then packed with gauze. The palate is now sutured in two layers; the dorsal layer is closed by interrupted catgut sutures, the ventral layer, by silk. Two silk mattress tension sutures are used. If the external carotid is not tied, the ligature is left in place for 24 hours after the packing has been removed.

My first patient was a boy of 16 sent from a mountainous part of the state where there was no medical practitioner for many miles. His chief complaint was frequent nasal hemorrhage, two of them severe enough to require transfusions. Examination showed the tumor mass filling the entire postnasal space and extending into the left naris. For several reasons it was decided to approach this surgically through the palate, as this appeared to be the most direct route to the mass. I was further influenced by observing on several occasions the satisfactory repair work on the palate by one of my colleagues.

The next patient, a man of 29, who gave a history of nasopharyngeal symptoms of more than ten years' duration, was referred on account of the presence of a mass in the pharynx, occasional nasal hemorrhage, and difficult deglutition. He had been living on a milk diet for two months, and it was approaching the point where even milk was difficult to swallow. He had lost 30 pounds in three months. Examination showed the tumor filling the entire nasopharynx down to the hypopharynx. At operation, the origin was on the left side of the base of the skull, and the growth extended into the left naris. The tumor measured 12 cm. by 4 cm. after removal.

A boy of 16 was admitted to the hospital for nasal hemorrhage which could not be controlled by packing. Examination revealed a large fibroma. As an emergency measure, the right external carotid was ligated, promptly stopping the hemorrhage. A transfusion was given. Later the growth was removed in the usual manner and was found to originate from both sides of the base of the skull. On removal of the mass from the left side, the bleeding was so severe that it was necessary to tie the left external carotid in order to control the hemorrhage.

The other three cases were without incident.

SUMMARY

The tumors were large in all of the cases, and, in the majority, filled the postnasal space. All had nasal extensions. Five patients were referred because of severe hemorrhage. Preoperative transfusions were necessary in three cases. No postoperative transfusions were required. The ages of the patients were as follows: 13, 16, 16, 19, 28, and 29. All were males. One boy of 16 had a recurrence in seven months with severe bleeding, as he had had before the first operation. The same approach was used, and a very hard, small, fibrous mass was removed from the original site. It was apparent that a small base of this mass was missed at the first operation. There has been no recurrence in six years in this case.

All the patients recovered. There was one recurrence. The last patient was operated upon three years ago. All tumors were diagnosed microscopically before or afterward or both. The period of hospitalization averaged ten days.

CONCLUSIONS

In the treatment of nasopharyngeal fibromata there are two main therapeutic measures: radium and diathermy, or surgery. If

one chooses surgery, it is believed that the transpalatine route offers the best opportunity for successful removal. The preliminary ligature around the external carotid adds considerable security to the procedure. The double line of sutures in the palate, plus chemotherapy, prevents perforation. We believe the transpalatine approach has a definite place in any consideration of the treatment of this type of tumor.

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XXXII

INFLUENCE OF CONSTITUTIONAL FACTORS IN OTOLOGICAL CONDITIONS

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While our concepts of otological conditions in the past few years have been guided and frequently altered by the advances made in the fields of physics and electronics, as applied to the immediate transmission of sound to the sensorium, the local impeding factors which may modify it and the operative procedures devised to facilitate this transmission, it is noteworthy that certain constitutional factors have not been given the proper consideration as contributory elements in the causation of many of these otological conditions. These conditions may be the result of actual local pathological, as well as physiopathological states which have given rise to functional changes. Neither time nor the present circumstances warrant an exhaustive consideration of the clinical otopathies which may accompany syphilis, tuberculosis, diabetes or other states of general debility.

The science of geriatrics points to the fact that changes occur in the tissues of the body as a necessary accompaniment of inevitable senescence. These are the result of antecedent changes in the glands governing function whereby their secretion is reduced below a point necessary for the proper maintenance of the integrity and function of body tissues. In certain endocrinopathies the function of these glands is so interfered with that changes in special tissues or organs are prematurely precipitated.

It is not our problem to determine the causes of dysfunctions of the thyroid, the parathyroid, the pituitary or the adrenal glands, though it is distinctly our concern as otologists to be cognizant of the effects of these dysfunctions. The local resistance of the mucosa of the eustachian tube and of the middle ear is dependent upon the integrity of these tissues. This lack of integrity or this susceptibility to infection is in turn dependent upon the state of the epithelium and

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of the subepithelial tissues. The ciliated epithelium which is undergoing albuminoid changes cannot function and cannot prevent the penetration of bacteria into the subepithelial tissues which are likewise in a myxedematous state as a result of hypothyroidism. This condition of the eustachian tube is in itself frequently sufficient to interfere with the proper opening of the tube and the ventilation of the middle ear and results in varying degrees of conduction deafness. The myxedema may likewise involve the entire mucosa of the middle ear, thus intensifying the deafness.

It is obvious, therefore, that treament of this type of tubal obstruction by simple inflation will be of no avail in relieving the deafness and the accompanying symptom of fullness in the ears, unless supplemented by adequate thyroid therapy. Such a therapeutic regime should not be undertaken empirically, but should be guided by initial and subsequent basal metabolic studies. This phase of investigation is within the province of the careful, practical otologist. It is an accepted fact that hypothyroidism is frequently a cause of lowered resistance to infection and it should be recognized as such and as a contributing factor in aural infections.

The perception factor is likewise important in impaired hearing in hypothyroidism. Whether this perception deafness exists because of the innate mental dullness of the patient, a hydrops of the cochlea or auditory nerve, or an actual albuminoid effusion into the nuclear centers, is conjectural. That there is an interference in reception is undeniable, since deafmutism is commonly found in cretins.

Audiometric studies corroborate the fact that such deafness in otherwise normal subjects is definitely benefitted by thryoid therapy and will tend to recur if therapy is not persisted in. The complete restoration of hearing, however, does not necessarily follow, since in many cases there remain residual elements of the albuminoid effusion in vital pathways. We have seen cases in which the hearing has improved without an appreciable improvement in the audiogram. This we have attributed to the subjective improvement in the sensorium and in the general health of the patient as a result of thyroid therapy.

Tinnitus may occur in hypothyroidism, with or without an impairment in hearing. This may be explainable on the basis of the lowered blood pressure and the atonic state of the large vessels and tissues adjacent to the middle ear and the eustachian tube, or it may be the result of an albuminoid distention of the cochlear cells.

As the clinical condition of the patient improves, relief may be obtained.

Allergic edema of the mucosa of the eustachian tube or of the middle ear, or both, will give rise to unilateral or bilateral conduction deafness, with or without a sterile serous otitis media. This is associated more often with ingested substances than with inhalants or pollens. The severity and the duration are in proportion to the sensitivity of the patient. Offending allergens should be sought for and the usual routine measures of avoidance of these allergens, specific desensitization, and administration of specific drugs to counteract the allergic state, should be pursued. In Ménière's syndrome the possibility of an allergic etiology should always be considered. Also, certain types of diffuse otitis externa may be the result of an allergic background. They are usually associated with similar manifestations elsewhere about the body and occur more frequently in infants and young children. Such conditions are notoriously resistant to treatment even after the removal of the alleged allergens.

While vitamin A is referred to as the anti-infectious vitamin, the evidence is conflicting as to its ability to act as a prophylactic or curative agent in general infective processes. Consequently we do not feel that its presence or absence has any specific effect upon the prevention of the predisposition to otitis media or mastoiditis, per se. It is said to influence calcium metabolism.

The general acceptance of vitamin B (thiamine) as the anti-neuritic vitamin is based upon its curative effect in polyneuritis, specifically in beriberi which is an inflammation of the peripheral nerves. From this fact has developed the practice of using thiamine in the treatment of receptive deafness. Physiopathologically the conditions are not analagous and it is illogical to expect similar benefit to the auditory nerve or its endings. This has been corroborated by the clinical experience of many of us.

The principal action of vitamin D is the promotion of the deposit of calcium and phosphorus in bone, and as such it is indispensable in the human economy. In otological conditions, the deficiency of this vitamin would retard the rate of the deposit of calcium and phosphorus following mastoidectomy, while its presence in normal amounts would be a contributory factor in the deposit of these salts in otosclerosis.

The lowering of resistance to infections of the upper respiratory tract that is attributed to specific vitamins is more likely the result of a marked deficiency, not to one, but to many vitamins. This

becomes aggravated by the infections themselves which are known to cause vitamin depletions. Thus the replacement of such vitamins together with the correction of other constitutional deficiencies will help to overcome aural and mastoid infections, and if any benefit in hearing is derived from their administration, it is the result of the improvement in the general health and alertness of the patient.

The parathyroid gland, because of the influence which its hormone exerts upon the distribution of calcium, phosphorus and magnesium in the tissues and the fluids of the body, assumes a position of great importance to the otologist. This parathyroid function is undoubtedly governed to some extent by the influence of the anterior lobe of the pituitary. An extreme instance of hypofunction occurs in Cushing's disease or pituitary basophilism. Among other constitutional changes there is the loss of mineral from the bones, with softening or brittleness. However, Susman[1] found that hyaline degeneration of these cells was invariably present, without the general increase in basophile cells. This implies a modified hypopituitarism which could so effect the parathyroid function that a disturbance in the mineral salt balance between the body fluids and the bones could take place, without giving rise to the more apparent symptoms of hypopituitarism.

The maintenance of the proper balance between the calcium intake and the plasma and bone calcium and phosphorus is essential to the proper functioning of the entire organism and the avoidance of certain pathological bone changes. The normal calcium content of the blood serum is between 9 and 11.5 mg. per 100 cc. while in bone, calcium makes up 15 to 18 per cent of the weight of fresh tissue and the ratio of total calcium to phosphorus is 2.2:1, or 10 atoms of calcium to 6 of phosphorus. Best and Taylor[2] state that "It is now generally believed that the calcium carbonate and calcium phosphate of bone are present not as separate compounds simply mixed together with smaller amounts of other mineral salts, but as a complex compound." It is well known that the calcium-phosphorus balance is reduced, or negative, in infantile rickets, osteomalacia, hyperparathyroidism, hyperthyroidism and after a period of growth, especially in women after puberty, pregnancy and lactation. However, during the period of growth and pregnancy, in acromegaly, or after a period of calcium starvation there occurs a calcium retention and the calcium balance is positive.

A hypercalcemia or a hyperphosphatemia is as a rule pathognomonic of a depletion of bone calcium. This may be caused by

a parathyroid tumor, a metabolic depression of parathyroid secretion, or the excessive or prolonged administration of vitamin D, parathormone or irradiated ergosterol, all of which cause a withdrawal of calcium from the bone.

According to Robison[3] phosphatase is an enzyme present in bone, capable of hydrolyzing various phosphoric esters and primarily responsible for calcification. It is present in greatest amounts in ossifying cartilage, in smaller amounts in formed bone, but is absent from resting epiphyseal cartilage and from nonossifying cartilage in other situations, though bone is apparently the main if not the sole source of plasma phosphatase. Phosphatase is increased in osteomyelitis, fragilitas ossium, acromegaly, infantile, renal and adolescent rickets, osteitis fibrosa cystica and osteitis deformans.

The explanation of calcification with which we are vitally concerned in the repair of bony defects following mastoid surgery, in the causation of otosclerosis and in the ossification of the fenestra following the fenestration operation for the relief of otosclerosis, does not rest on any one factor. Rather, there are several simultaneously acting factors which no doubt are responsible. The three most plausible explanations are: the precipitation of calcium salts from a supersaturated solution in body fluids by ionization; the active secretion by the osteoblasts of bone salts derived from the calcium and phosphorus of the blood; the action of phosphatase and vitamin D.

While the foregoing physiochemical theories and facts are accepted in our knowledge of the changes occurring in pathological conditions of long bones, it is difficult to apply them in our search for an explanation of the physiopathological changes in the otic capsule in otosclerosis. There are factors which activate the ebb and flow of the mineral salts between the body fluids and the bone as a result of which these bone changes are initiated, continue to progress or undergo regression, but which apparently do not affect the otic capsule. On the contrary, once initiated, the changes in the otic capsule are progressive. The rate of progression varies in different individuals and at certain times in life. During pregnancy, for instance, the calcium and phosphorus content of the bone and body fluids is increased, while after pregnancy it is decreased. Following the pregnancy of an individual in whom the predisposition to otosclerosis exists, the depletion of bone calcium and phosphorus may activate the otosclerotic focus because of the disturbed local metabolism as influenced by the estrogenic imbalance. Whereas, after

this postpartum period, when the calcium balance changes from a negative to a positive phase, the solubility product constant for the precipitation of calcium and phosphorus salts is exceeded, the excess calcium and phosphorus continues to be deposited in the otosclerotic area as a result of the increased activity of the focus, and the clinical evidence of otosclerosis becomes more firmly established.

It is probable that phosphatase also takes part in this process since it is stimulated by the growth of bone and by diseased bone, and is increased in fragilitis ossium which has been found to coexist with otosclerosis. It is also present in greatest amounts in ossifying cartilage and may be the chief factor in the laying down of calcium in the cartilaginous rim of the fenestra ovalis, leading to fixation of the stapes.

There is thus very little accurate data on which we can establish a connection between constitutional dyscrasias of bone and the changes in the otic capsule in otosclerosis. There are certain constitutional physiochemical changes, however, which strongly suggest that the interaction of the secretions of endocrine glands with the metabolism of the mineral salts may have a significant bearing on the progress of otosclerosis.

It would, therefore, seem reasonable to consider the hypothesis that the changes that occur in the capsule are in the nature of a local metabolic disturbance of osteogenic physiology, which has its incidence in the age group in which certain abnormal cytologic bone changes occur, which may overstimulate the normal processes of repair of diseased bone, namely fibrosis and calcification causing the precipitation of calcium and phosphorus by ionization and by the actions of phosphatase and vitamin D in individuals who are predisposed to otosclerosis.

It is, therefore, apparent that otological conditions are not the result of local tissue changes only, but may be manifestations of profound constitutional variations in the physiochemistry of the patient. The appreciation of this fact should direct our attention to the more thorough analysis of the patient as a whole in order to afford greater relief and to anticipate recurrences before permanent irreversible changes become established.

CONCLUSIONS

1. Hypothyroidism and allergy may cause deafness, tinnitus, vertigo and serous otitis media.

2. Deficiency in vitamins A and B may contribute to general lowered resistance and predisposition to infections of the middle ear.

3. Disturbances of function in the pituitary and parathyroid glands and deficiency in vitamin D may give rise to faulty metabolism and distribution of calcium and phosphorus leading to abnormal bone changes in the otic capsule.

4. It is suggested that the changes which occur in the otic capsule in otosclerosis may be the result of local metabolic disturbances in osteogenic physiology and the processes of bone repair.

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XXXIII

DIVERTICULUM OF THE ESOPHAGUS

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As an introduction to this subject, I should like to philosophize briefly on the present status of otolaryngology and its opportunities for the future, to allude to a pervasive expression of discouragement and to offer a suggestion for the solution of a problem that seems to be causing apprehension among our fellows in the profession. A most competent and skillful colleague recently stated, that as far as his practice was concerned, a training period of six months after the internship would have been sufficient to equip him for his daily tasks. Specifically, he and many other members of the otolaryngological profession are deeply concerned with the alleged curtailment of practice resulting from the introduction of the chemotherapeutic agents and antibiotics in the treatment of acute infections. They point to the almost complete annihilation of temporal bone surgery and the procedures for the relief of the complications of mastoiditis, the marked decrease in the incidence of acute suppurative processes in the mouth, pharynx, and cervical region, and the rare occurrence of the serious sequelae of acute nasal accessory sinus disease. These conditions obviously cover a broad field and their rarity today may in some measure justify the conviction that otolaryngology has entered a period of decadence.

Let us pause for a moment to take stock of this situation, to analyze the present possibilities and future potentialities of our specialty, and to give ourselves, if possible, the right answers. A casual survey of the field of otolaryngology would seem to indicate that there occur many diseases of the head and neck which should attract the attention of the otolaryngologist and justifiably fall within the province of his practice. Consider, for example, diseases of the salivary glands, particularly the tumors, neoplasms about the head and neck, faciomaxillary plastic surgery, operative procedures for the improvement of hearing in otosclerosis, and a host of others, all offering challenge to the skill and talents of the members of our profession.

Immediately it will be said that these conditions are irretrievably lost to us because they are now in the hands of the general surgeon.

But why alone in his domain? Not because the general surgeon displays unusual competence or skill in dealing with these afflictions, but largely for the reason that he has been trained in a more precise and comprehensive manner in the principles of surgery, the factors which favor tissue repair, and the preoperative and postoperative care of the patient. Without meaning to detract in the slightest from his efficiency and capabilities, I venture to state that many of the operative procedures about the head and neck can be dealt with more skillfully from a technical standpoint by the otolaryngologist than by the general surgeon. The former's training in delicate techniques, his skill in using direct and reflected light, and his knowledge of the anatomy of these parts should make him well prepared and equipped to deal successfully with these conditions.

But before the otolaryngologist embarks upon this broader field, there are certain educational responsibilities which he must assume. He must be thoroughly trained in the principles of surgery, newer surgical techniques, protein metabolism, the value of early ambulation, the recognition and treatment of shock, methods of resuscitation, and the use of modern anesthetics, if he is to practice surgery of the head and neck with credit to himself and safety to his patients. He need not deal with these technical and biochemical problems in too specific a manner or in too large quantities to prepare himself adequately for the broader tasks which invite his attention. I dare say that two months of experience and observation in a teaching department of general surgery will suffice to equip him with the necessary knowledge of these practical considerations, provided his early education has given him a good grasp of medicine and an adequate training in the techniques pertaining to his special field.

There is still another great resource which has not been tapped by the otolaryngological profession, at least not to any appreciable extent in highly specialized fields, namely, scientific research. As far as I am aware, there is but little significant basic and fundamental research in otolaryngology anywhere today in this country. Yet, we are fully aware that where there is no research there can be no progress in medicine. We must have members of our profession who observe and envision opportunities for study, men who are capable of promoting theories and reducing them to facts by careful clinical and laboratory experimentation. It behooves us, also, to recognize these facts when they are available, to appraise them promptly and to put them into practical usage when they possess something of value in the care of the sick. It is a sad reproach to the scientific world that many of our greatest discoveries have gone

unnoticed or receive only scant attention for a decade or longer after they were made available to us. Sulfanilamide was prepared in 1908, yet it remained dusty in organic laboratories for a quarter of a century before it was realized to be a great savior of suffering and of life. Penicillin was discovered in 1929, yet it remained obscure and attracted little attention until the advent of World War II.

There are colossal opportunities for research in otolaryngology, the products of which will open many new fields for practice. I need only mention the problems of deafness, the rehabilitation of the hard of hearing, nerve regeneration, the neurological manifestations of otolaryngological diseases, and the common head cold to prove that there are many investigative studies in which reputations may be made and from which newer methods and procedures of practice will be derived.

Creativeness is the greatest thing in life. What a joy and pleasure it is to invent some new instrument that will change the surgical failures of the past into successes of the future, or to discover some new therapeutic agent or method of treatment that will prevent disease or minimize human suffering. And what enviable opportunities we in medicine encounter daily for these achievements. Let us not fail in training our young men in otolaryngology to build a strong basic educational foundation upon which they may erect a superstructure of such unlimited expanse that they will never be given to an expression of futility and despair—that otolaryngology in its decadence has finally led them into a blind alley.

We come now to the text of my discussion, diverticulum of the esophagus. I should like to make a plea for the one-stage operation, emphasize a few technical features of this procedure, and allay, if possible, some existing apprehension concerning its dangers.

In regard to etiology, there is little that can be said which is new. A diverticulum may occur in any part of the esophagus, although the lesion to which reference is now made is found posterior to the cricoid cartilage at the junction of the hypopharynx and the esophagus. It is classified as a pulsion diverticulum in contrast to the traction diverticulum which is usually seen in the middle third of the esophagus. Because of its location it is frequently termed a pharyngo-esophageal sac.

No one has yet posed a universally accepted explanation for the cause of these lesions. There is some support for the theory that they are congenital, yet we rarely find them occurring in more than

one member of a family. In favor of a congenital malformation is the uniform site of the sac, between the lower fibers of the inferior constrictor muscle of the pharynx and the cricopharyngeus muscle, in a region where it is assumed some irregularity or disturbance of embryonic development has produced a muscular weakness.

There exists, however, another influence which has been present in a number of my cases and perhaps has passed unrecognized in others, namely, an esophageal obstruction. Such a condition may often be the exciting factor, particularly when associated with a congenitally weakened tissue resulting from muscular deficiency in the region above described. In a patient recently seen in the clinic suffering from ulcerations of the upper part of the esophagus, with consequent obstruction, we watched the development of a typical pharyngo-esophageal diverticulum. It is perhaps reasonable to assume that the abnormal strain and pressure of the swallowing effort exerted upon the region between the inferior constrictor and the cricopharyngeus muscles were the exciting factors in the production of a herniation of large enough proportions to extend into the posterior mediastinum. In every instance of pulsion diverticulum, we should search for an underlying esophageal obstruction and remedy this condition, if possible, after excision of the esophageal sac. A persistent esophageal lesion producing some degree of obstruction may be responsible for the rather frequent recurrences of esophageal diverticula after what appeared to be successful operations for their removal.

The first symptom of pharyngo-esophageal diverticulum is usually that of obstruction. The patient states that food lodges in his throat and that it sometimes causes him to have a choking sensation or a feeling of suffocation. He often complains of a gurgling sound in his throat during the act of swallowing and later the regurgitation of food when the sacculation has become large enough to hold a considerable portion of each meal. The patient may find relief from the feeling of a foreign body in his throat by compressing the side of the neck and milking out the contents of the sac. Large diverticula may compress the esophagus and cause complete esophageal obstruction. In these instances the patients often tell you that they have no difficulty in swallowing the first part of their meal but experience considerable obstruction before the entire meal has been ingested. As the large sac fills, it compresses more and more upon the esophagus and may ultimately produce a degree of obstruction that renders the ingestion of liquids difficult or even impossible. Very large sacs extending into the posterior mediastinum may pro-

duce some respiratory difficulty or cause embarrassment to cardiac action. Such lesions occasionally exert sufficient pressure upon the recurrent laryngeal nerves to produce a paralysis of one of the vocal cords. The symptomatology of esophageal diverticulum may be somewhat vague in the early stages of the disease, but later, with enlargement of the sac, the diagnosis can usually be made from the patient's history. The symptoms then become definite and of such a specific character as to leave little doubt of the true nature of the disability. A roentgenogram can be expected to reveal the lesion, its extent, and its precise location.

Recent progress in surgery has indicated the extreme importance of paying meticulous attention to the details of preoperative care. The preparation of the patient has much to do with the manner in which he tolerates a surgical insult and his behavior during the period of convalescence. In this connection too much emphasis cannot be placed upon the protein requirements of the patient. The degree and rate of tissue repair are tremendously influenced by the amino acids, and any patient who is the victim of their impoverishment is destined to react poorly to a major surgical procedure.

In no other class of patients are we likely to find a protein deficiency more often and to a greater degree than in those individuals who have been afflicted with a prolonged esophageal obstruction. Starvation has been their chief detriment; a weight loss of considerable proportions is usually recorded and a low protein content of the blood is certain to be present. The surest evidence of this form of malnutrition is a major loss in weight. Laboratory tests for the protein content of the blood are available but frequently unreliable in those patients where the total volume of blood is reduced. The protein concentration of the blood may be normal, but if there is a decrease in blood volume, the total content of protein in the patient may be entirely inadequate. In every patient who gives the history of an inordinate weight loss over a period of 30 to 90 days, one must expect a hypoproteinemia.

It is exceedingly important, therefore, to supply adequately the protein requirements of the patient before surgical procedures are instituted. This can be done by using an indwelling esophageal tube, intravenously, or in some cases perhaps, by performing a gastrostomy. Amogin may be administered intravenously, 100 gm. daily for several days preceding and after the operation. It must be remembered, however, that no form of feeding takes the place of ingestion. Milk, eggs, and meat given by mouth are much more

effective than the administration of their food values by any other method. It has been estimated that proteins taken into the stomach are twice as effective as those given intravenously. The extreme importance and value of an adequate blood protein in the patient operated upon for the removal of an esophageal sac is reflected in the fine manner in which the wound repairs when this requirement has been given the respect which it commands.

The one-stage operation is strongly advocated. It shortens the period of convalescence and, therefore, obviously lessens the expense of illness and in my experience seems less likely to be followed by a recurrence of the lesion. The two-stage operation quite properly had its advocates for a number of years because it was a safeguard against the possible development of a suppurative mediastinitis. It offered little risk of exposing the mediastinum to the infected contents of the sac and, therefore, reflected a better mortality record for many years than did the one-stage procedure. Newer technical methods of closing the pedicle of the diverticulum and the advent of the chemotherapeutic and antibiotic agents have now rendered the one-stage operation comparatively safe and perhaps also the surgical procedure of choice today.

There is little doubt that sulfadiazine and penicillin have greatly reduced the incidence and the mortality of mediastinal infections. An increasing number of reports of acute suppurative mediastinitis secondary to foreign body perforation of the esophagus, cured by the administration of the chemotherapeutic and antibiotic agents, are coming to light wherever this subject is discussed in our medical meetings. It is not unusual to hear one of our colleagues recount an instance of mediastinal infection due to foreign body perforation of the esophagus with resolution of the process without surgical interference after the administration of sulfadiazine and penicillin. Even in those cases where there is a leak in the esophagus after excision of the sac, there need be less apprehension than formerly concerning the dangers of a mediastinitis because of the present-day therapeutic armamentarium at our disposal. It is somewhat consoling to observe that most esophageal fistulae occur three or four days after the removal of the diverticulum. It is highly probable that, in those patients in whom this undesirable sequel occurs, the fascial planes leading to the mediastinum have been sealed before the contents of the esophagus are drained into the soft tissues.

A good roentgenogram will serve to indicate the precise location of the sac. It is usually found on the left side but in some

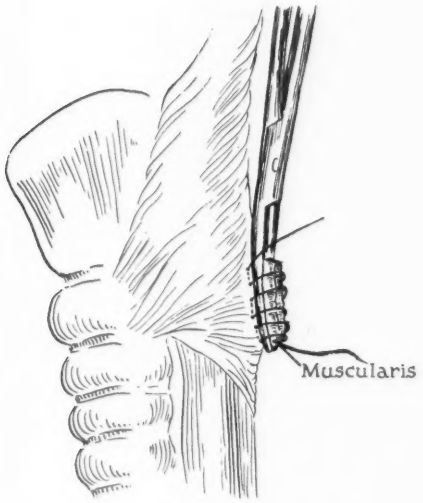

Fig. 1.—Cushing Suture. A continuous mattress stitch which does not enter the lumen of the pedicle and thus avoids contamination of needle and suture.

instances it may be seen more to the right. The approach to the diverticulum, therefore, is on the side of the herniation. If the sac is directly posterior to the esophagus and there seems to be little choice in the site of approach, one will usually find it somewhat easier to reach the diverticulum from the left side because of the slight normal deviation of the esophagus to the left of the midline of the neck.

Under local anesthesia, with the patient deeply narcotized, an incision is made through the skin, the subcutaneous tissue, and the platysma myoides muscle, along the anterior border of the sternocleidomastoid muscle from the level of the upper border of the thyroid cartilage above to a point two or three centimeters above the suprasternal notch. The line of cleavage between the sternocleidomastoid muscle and the omohyoid and sternohyoid muscles is identified, these structures separated, and the latter muscles retracted toward the midline. The thyroid gland is then exposed and its inferior pole freed and reflected upward, thus stretching the pretrachial fascia and facilitating its identity over the trachea and the esophagus. The pretrachial fascia surrounds the thyroid gland,

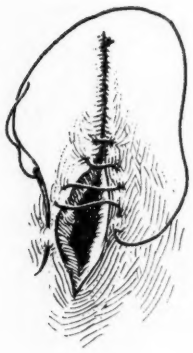

Fig. 2.—Sketch of a wound being closed by the Cushing suture. Note position of the needle in the superficial tissues.

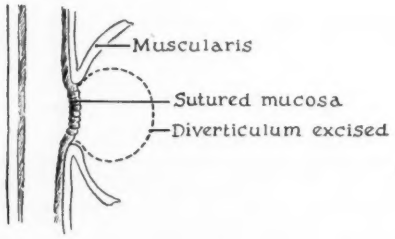

Fig. 3.—Lateral view showing closed pedicle of mucous membrane. The muscular cuff is then sutured over the closed pedicle.

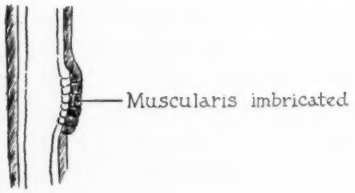

Fig. 4.—Lateral view of the muscular cuff sutured over the pedicle. Interrupted mattress stitches may be used to close the muscular cuff and bury the mucosal pedicle.

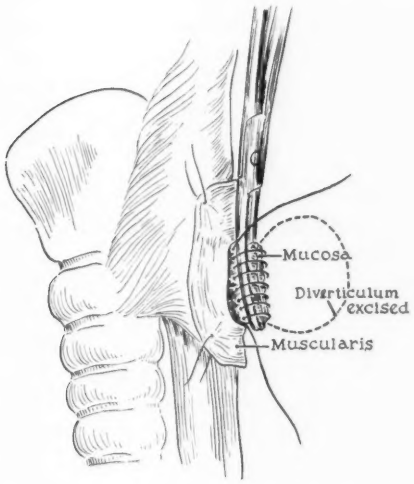

Fig. 5.—Continuous whip-over suture preferred by the author (lateral view) to close the pedicle. A purse string closure is effected while the clamp is being removed. The needle and the suture enter the lumen of the pedicle but the dangers from infection are minimal.

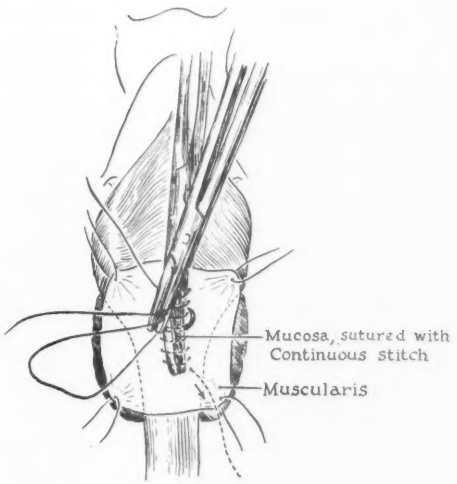

Fig. 6.—Posterior view of the continuous whip-over suture to close the pedicle.

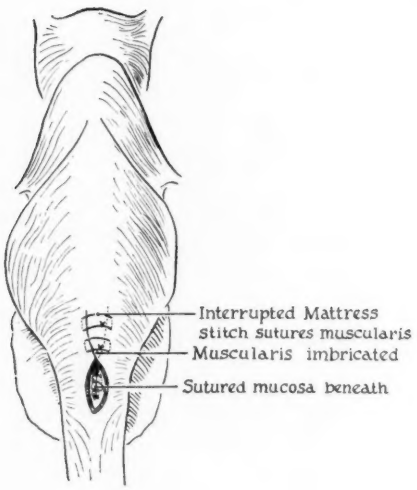

Fig. 7.—The muscularis is closed over the mucosal pedicle with interrupted mattress sutures. Interrupted single sutures may be used.

trachea, and esophagus, and forms the so-called visceral compartment of the neck. When incised, it allows a direct approach to the diverticulum, which is situated approximately at the level of the cricoid cartilage. Large diverticula are easily seen but the smaller ones, especially those originating from the posterolateral wall of the pharynx, are sometimes difficult to locate. If the lesion is not readily identified, the patient is asked to swallow sufficient water to dilate the sac. The reflex act of swallowing will be maintained, even in those patients who are deeply under the influence of narcotics.

When the sac has been found, it is carefully separated from surrounding fascia, grasped with Babcock forceps, and raised slowly into the neck wound. Care should be taken to free all the fascial coverings of the sac and to avoid perforation while one is attempting to draw out the pedicle to its smallest dimension. It is not covered by a distinct muscular layer, although muscle fibers of the inferior constrictor and of the cricopharyngeus muscles are usually found about the neck of the sac and must be separated in order to expose the true pedicle.

When the sac is entirely exposed, a Bard-Parker knife is passed gently around its dome in order to sever a thin layer of fascia or a serous coat, which is to be reflected downward toward the pedicle in the form of a cuff. The cuff can be improvised by carefully separating the fascial covering from the fundus of the sac by manipulation and retraction with gauze over the index finger. As the pedicle is approached the cuff becomes thicker and finally of a substantial fabric when the fibers of the inferior constrictor and cricopharyngeus muscles are reached. The preservation of this cuff is exceedingly important to the closure of the pedicle without leakage in the final stages of the operation.

A Pryor clamp or a moderately curved hemostat with longitudinal treads and without teeth is now applied to the pedicle within the muscular cuff and the sac is excised lateral to the instrument. The infected, cut surface is then treated with phenol and carbolic acid. At this point two methods of suturing the pedicle may be employed, the Cushing suture which when properly executed does not enter the lumen of the pedicle, or the whipover suture which ultimately closes the neck by the purse-string method.

The Cushing suture is a continuous mattress suture used in such a manner as to avoid the interior of the pedicle and contamination of the needle and the suture material (Figs. 1, 2, 3 and 4). I prefer the continuous over-and-over closing suture (Figs. 5, 6 and 7), because of the purse-string hitch which it executes, thus reinforcing a region in which there is always the possibility of a recurrence of the sac. Finally, when the pedicle has been closed, the muscular cuff is imbricated with interrupted single sutures or mattress sutures to bury the stump. I prefer absorbable suture material, No. 00 plain catgut on a swedged needle.

CONCLUSIONS

1. It is urged that the otolaryngologist widen his scope of interest in the field of surgery. To this end he must also broaden his knowledge of the principles of surgery and modern preoperative and postoperative care of the patient.

2. A plea is made for greater activity in otolaryngological research. Scientific investigations speak for the progress which advances the boundaries of opportunities for practice.

3. Pharyngo-esophageal diverticulum is a herniation of mucous membrane through an alleged weakened region at the junction of

the inferior constrictor and cricopharyngeus muscles. It can be successfully removed with a one-stage operation, the advantages of which in terms of the period of disability and expense are obvious.

4. While we must not relent in our effort to give meticulous attention to the technical details of the operation, we need not be greatly alarmed by the appearance of an esophageal fistula. Sulfadiazine and penicillin used postoperatively have greatly reduced the dangers of this complication.

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XXXIV

ENDAURAL SURGERY FOR CHRONIC MASTOIDITIS

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It must have been the experience of all otologists who have learned the technique of endaural surgery of the temporal bone in recent years to have a period of well-marked, early enthusiasm for the method. This occurs because in learning the technique under competent instruction and in seeing it applied in its many uses the novice becomes greatly stimulated by the new knowledge he gains. The endaural approach not only demands techniques that are for the most part completely new to the ordinary otologist but, in his study of the method, he finds that he has to relearn the entire anatomy of the temporal bone. The approach and the technique of performing the various steps of the many operations of endaural surgery allow him to see the structures in their normal relations from a point of view never before experienced by most students. His newly acquired knowledge in both anatomy and surgical technique seems, and indeed truly is, a revelation; to those who put their new skill to clinical use, the method may fairly be said to open a gate to a new and better period in surgical otology.

When this early enthusiasm has been tempered, not to say sobered by experience gained in putting the method to use by operating on the living patient and in the presence of pathologic changes as well as the normal anatomical variations that offer difficulties and handicaps in the work, there still exists an abiding feeling among the otologists who are carrying on with the method that it offers many real advantages. Not only the endaural approach but the whole method of surgical technique developed by Lempert, Shambaugh, Meltzer, and others deserves the general attention of all otologists.

The advantages of endaural surgery in chronic mastoiditis are numerous, and entirely realistic and of practical importance. Let us consider them in some detail.

Presented before the American Laryngological, Rhinological and Otological Society, St. Louis, Mo., April 26, 1947.

1. The endaural approach offers the most direct possible avenue of surgical attack on the external auditory canal, the mastoid antrum and mastoid cells, the aditus and tympanic attic, the zygomatic area, the tympanum and its boundaries, the semicircular canals and vestibule, the cochlea, and the petrous apex through both the anterior and posterior portions of the base of the pars petrosa. It also affords a ready approach to the lateral sinus, the dura of the middle and posterior cranial fossae, and the facial nerve in its entire course.

2. Endaural surgery can be performed under either local anesthesia with heavy premedication, or any suitable general anesthesia.

3. Bleeding in the endaural incision is usually less than that in the postauricular, and it is readily controlled by electrocoagulation.

4. The essential landmarks in each step of the various operations are exposed in turn and are fully visible as guides to the following steps of the technique. This visibility and direct accessibility when bleeding is controlled and the field kept clean by saline irrigation and suction are very great advantages over the postaural method; they are excellent at all times; with proper exposure and suitable retraction the auricle and the soft parts are not in the way at any time.

5. The direct accessibility and the good visibility enable the operator to examine both the anatomy and the changes produced by disease in minute fashion and to apply the most refined technique in handling the structures. Thus in the course of the modified radical mastoidectomy, the tympanum, the tympanic attic and the ossicles may be minutely examined. As a result of the findings, and because of the possibility of the most meticulous dissection that may be made, it is sometimes possible to perform the modified instead of the full radical operation that would have been deemed necessary by other methods; the additional loss of hearing usually noted after complete radical mastoidectomy is thus avoided. This preservation of function of the auditory organ is a pronounced feature of the technique. When the full radical operation is being done, the treatment of the tympanum can be most exact, with preservation of the stapes and round window areas, while at the same time disease in the auditory tube, the peritubal cells, and the hypotympanum can be most easily reached and eradicated.

6. There is minimal trauma to the soft parts, with little postoperative reaction, and infections such as erysipelas or cellulitis are very rarely seen.

7. There is no damage to the sternomastoid muscle attachment at the tip of the mastoid process; there is no sore neck postoperatively and descending infection of the neck must be very rare. There should be no serious damage to the temporal muscle.

8. Since the endaural incisions do not at any point cut into the cartilage of the pinna, perichondritis following operation can scarcely occur.

9. Postoperative drainage is perfectly free and direct with the wound left wide open, and retention of secretions cannot occur.

10. The interior of the operative cavity remains visible and accessible throughout the healing period. It is under complete control at all times through the endaural window which is always larger than the meatus created by the canal plastic of the postauricular technic.

11. After-care is very simple indeed and dressings are not painful.

12. The period of hospitalization, especially for elective surgery for chronic mastoiditis, is very much shortened; most of the patients can be allowed out of bed in 24 hours and can be sent home in a day or two if necessary. Since only a small dressing in the canal is necessary after the first week, there can be a very early return to work and to social activities. The period of disability is thus very short indeed.

13. There is no possibility of an unsightly postauricular depression or scar, nor of a persistent postaural fistula. The cosmetic results of the endaural technique are usually exceedingly good; the meatus is slightly larger than preoperatively and the antauricular scar is often scarcely visible.

The disadvantages of the endaural approach are comparatively few and largely relative.

1. The method of approach and of performing operations through the apparently limited opening provided by the endaural window, using the technique developed so largely by Lempert, requires special training and a high order of surgical skill. After competent instruction, this skill must be brought to perfection by each surgeon through much practice on the cadaver. The technique must be most meticulous if the full advantages of the method are to be gained. Endaural surgery is not for the untrained, incompetent, rough or careless operator.

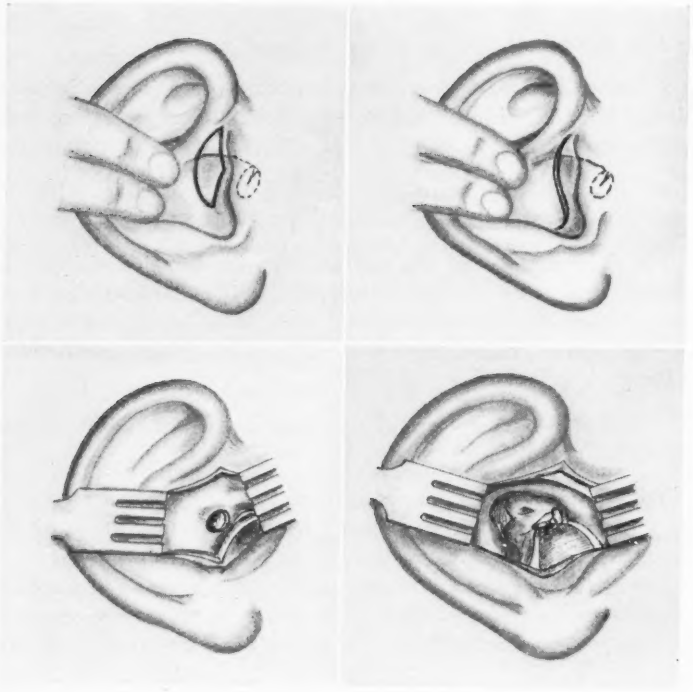

Fig. 1.—The endaural incisions of Lempert for the modified radical mastoidectomy.

Fig. 2.—The endaural incision of Shambaugh.

Fig. 3.—Exposure of the mastoid cortex and opening into the mastoid antrum.

Fig. 4.—Modified radical mastoidectomy with preservation of the ossicles, before cutting of membranous flap from canal.

2. While many of the steps of the technique can be performed by using the methods, equipment and instruments commonly employed in postauricular surgery, perfection of technique and results will only be obtained by the use of a considerable amount of highly specialized equipment and many new instruments, which even at this late day in the postwar period are not readily obtainable and are very costly.

3. Proper endaural surgery will only be possible when a trained and skilled operator has the required special facilities at hand.

4. Because of the somewhat limited size of the pathway of approach, there are certain technical difficulties which become less with growing experience and skill. It is not easy to reach, through the endaural window, mastoid cells which lie behind a superficial lateral sinus, or deep in a very large mastoid tip, or in the squama above the posterior part of an extensive mastoid development. It is not easy to expose the dura posterior to the lateral sinus unless the latter is fairly deep in a roomy mastoid cavity. Damage to the emissary vein within the posterior part of the mastoid cavity may be difficult to avoid and the ensuing hemorrhage may be difficult to control.

5. Despite the apparent small size of the window through which the operations are done, it is remarkable how accessible most of the structures become if the endaural incisions have been properly made, if the freeing of the soft parts has been adequate, if the retraction is skillfully done, all bleeding controlled, the wound kept perfectly clean by irrigation and suction, and the brilliant illumination of a suitable electric forehead lamp is employed.

Regarding considerations of technique, after a considerable trial of other methods, intratracheal intubation and the use of simple ether anesthesia has proven most useful. Light premedication, the usual gas-oxygen-ether sequence for induction, and intubation by the method of Flagg have proven quite satisfactory. It insures absence of pain, prevents shock, and avoids the occasional morphine poisoning noted with the heavy medication used with local anesthesia by some surgeons. There is little bleeding when the airway is always free, despite the position of the head. Venous congestion is avoided, oozing is minimized, and lengthy operations can be done with surprisingly light anesthesia. The patient is allowed to waken gradually and should be reacting mildly as the operation is finished.

The use of koagamin intramuscularly an hour before operation or intravenously during the performance may aid in slowing down

oozing. The bleeding from the initial incisions is lessened by the injection of procaine solution as if for local anesthesia. Bleeding is readily controlled by electrocoagulation.

The employment of the high speed, motor-driven dental engine to power the various perforating, cutting, and polishing burrs by which the greater part of the work on the bone may be accomplished through the small endaural window has revolutionized the technique. It is a speedy and labor-saving method; in trained hands it is an instrument of the most delicate precision when this is necessary. Damage can of course be done by the slipping of the whirling burr and it will cut into any tissue if it is misdirected; the skill of the operator will prevent the occurrence of such accidents. A further great advantage in the use of the burr over the mallet, chisel, gouge or rongeur is the absence of any concussion of the brain in patients in whom intracranial infection is suspected or actually present. Neurosurgeons have for years advised otologists not to use instruments of percussion in such cases; with the burr, all such effects are completely avoided.

The Modified Radical Mastoidectomy for Attic Perforation with Cholesteatoma. In general, the modified radical mastoidectomy with preservation of the drum and with or without preservation of the ossicles is indicated for attic perforations which usually have cholesteatoma within the attic and the mastoid antrum. By and large the operation is performed almost exactly as is the procedure of fenestration for otosclerosis; it thus affords excellent practice in this technique up to the point of making the fistula.

The endaural incisions employed are either those of the typical method of Lempert (Fig. 1) or the simpler incision of Shambaugh (Fig. 2); both afford excellent access. After the usual elevation of the soft parts with the auricle from the surface of the mastoid process, upward over the posterior root of the zygoma and beneath the temporal muscle, retractors are inserted. The cortex is penetrated immediately above and behind the spine of Henle with the cutting burr (Fig. 3); the bone down to the mastoid antrum is cut away by hugging the posterosuperior bony canal wall with the round cutting burr. Only enough of the heavy cortex is removed to give free access to the antrum, the aditus, and the posterosuperior bony canal wall, and to allow the removal of all disease and the inspection of the wall of the lateral sinus and the dural plate in both the posterior and middle fossae.

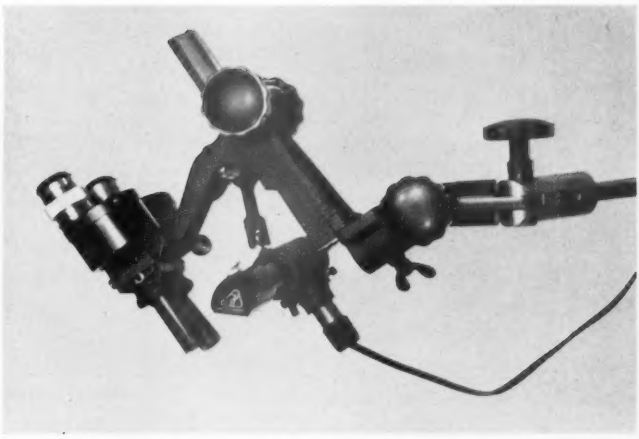

Fig. 5.—Ten power dissecting binocular microscope with modified Nichols illuminator.

The attic is now exposed by working forward over the top of the external bony canal; as always the horizontal semicircular canal which has been exposed on the inner wall of the aditus is the guide. The posterosuperior bony canal wall is thinned down, and after the membranous canal has been freed from it and preserved, the bony canal wall is taken down, the bridge over the posterior part of the notch of Rivinus is removed, and the facial ridge is lowered to its limit, which is absolutely flat with the horizontal semicircular canal (Fig. 4).

At this point a most careful inspection of the attic, the ossicles and the upper exposed portion of the middle ear is made with the loupe or Gullstrand spectacles, or preferably with the high magnification of the dissecting microscope of seven to ten power (Fig. 5). With good illumination and careful toilette by suction, the extent of the disease, which is usually cholesteatoma, granulation tissue or both, within the attic and the middle ear is determined, as well as any changes in the ossicles themselves.

As a result of this examination it will usually be found that with posterior attic perforations it will be possible to remove all disease from the attic and leave the ossicles in situ if they are normal.

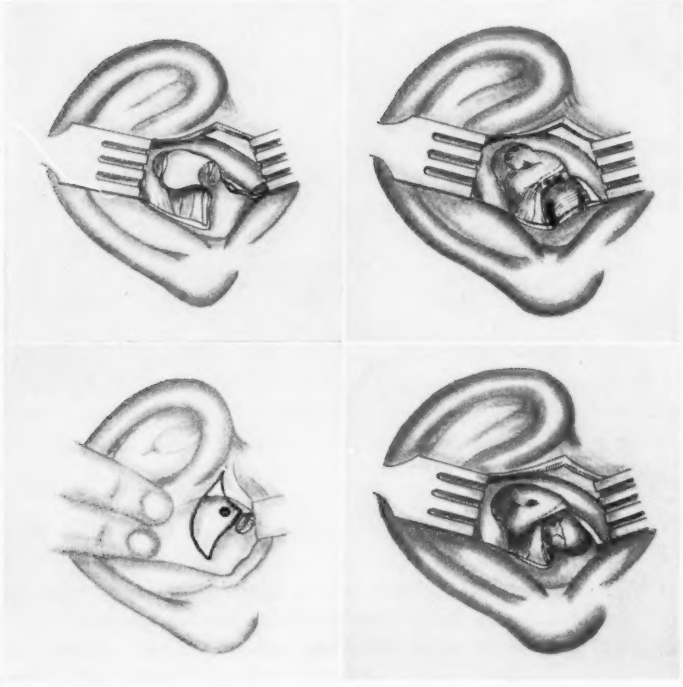

Fig. 6.—Completed modified radical mastoidectomy with membranous canal flap in place.

Fig. 7.—Modified radical mastoidectomy after removal of incus and head and neck of malleus; first incisions for cutting flap from membranous canal.

Fig. 8.—Incisions of Lempert for complete radical mastoidectomy. Point of initial opening of mastoid antrum through superior posterior bony canal wall.

Fig. 9.—Completed radical mastoidectomy.

A flap is cut from the membranous canal exactly as for fenestration (Fig. 6); it is applied over the ossicles by packing it gently against the inner wall of the attic, the aditus and the mastoid antrum with a gauze pledget, and then filling the cavity with moist sea sponge in a collar of paraffin mesh or rubber, after the technique of Shambaugh. The posterior attic perforation is thus obliterated by placing the membranous flap against the inner attic wall. The packing is completely removed in five or six days.

Anterior attic perforations with cholesteatoma usually require a different treatment; the head of the malleus and the body of the incus are in the way and prevent obliteration of the perforation against the inner attic wall. The incus and the head of the malleus are removed in the manner of the fenestration operation (Fig. 7). The flap is then cut and packed against the exposed inner attic wall after meticulous cleansing of the area of all disease, as described above. With disease of the ossicles, the same course will have to be taken by the removal of at least the incus and the head of the malleus.

The Complete Radical Mastoidectomy. In general, the full radical mastoid operation will be required for the cure of chronic otitis media with marginal perforations of the pars tensa of the drum, when there is cholesteatoma within the tympanum, the attic and the antrum. It is usually also imperative in exacerbations with impending or actual complications; this condition is still occasionally seen despite intensive chemotherapy.

The incisions used may be those of either Lempert or Shambaugh, as for fenestration, with the addition of the deep canal incisions of Lempert to expose the entire posterosuperior bony canal at once (Fig. 8). These are usually made with greater ease after the elevation of the soft parts and the insertion of retractors; visibility is better in the depths of the often narrow exernal canal; removal of the triangle of membranous canal thus outlined is easy.

The approach of Lempert through the bony canal wall at the junction of the inner and middle two thirds of a line drawn perpendicularly in from the spine of Henle to the drum margin with a small perforating burr allows of very quick and easy access to the mastoid antrum. Only a few millimeters of bone will have to be traversed, while approach through the cortex from the surface usually means cutting away two or three centimeters of ivory-hard bone. The method has the further very great advantage that it enters the antrum while avoiding the lateral sinus, no matter how far forward and superficial the latter may be, as the antrum is penetrated deep

to the sinus. In addition, a low-hanging dura in the tegmen antri is avoided since the antrum is entered well below it.

The opening into the antrum is quickly enlarged with the cutting burr to expose the horizontal semicircular canal and the aditus, which are the essential landmarks for further work. The technique from this point is the same as for the modified radical operation. The attic is opened widely, the rest of the bony canal wall is thinned down and removed with the bridge, and the ossicles or their remains, and the drum are all removed; the attic is then meticulously cleansed of all disease; the use of the magnifying loupe cannot be too strongly urged for this step.

The tympanum is next attacked; here again the great advantage of the endaural approach with its direct access and free visibility is of the utmost importance. Using the loupe, suction and irrigation and epinephrine pledgets for the control of bleeding, all gross disease is removed (Fig. 9). One then exposes and removes the cochleariform process and the tensor tympani muscle with its bony canal. The auditory tube mucosa is removed with any bony disease and peritubal cells that may exist. There is perfect access to the hypotympanum and the floor of the external canal can be lowered in a moment with the cutting burr; the overhang of the posterior tympanic space can be readily removed in the same way. Because of the good visibility the danger areas can be easily avoided; these are the facial nerve in its tympanic course and at the knee, the stapes in the oval window, and the niche of the round window. A final toilette of the tympanum and the attic under the dissecting microscope is well worth while.

There is no flap to be cut except the small remnants of the membranous canal above and below; the cavity is lightly packed for hemostasis. This packing is removed in five or six days; no further dressing except cotton in the meatus is required.

Granulations cover the exposed bone in the cavity; epithelization takes place from existing skin margins in four to six weeks. If quicker epithelization is desired, small Thiersch grafts can easily be inserted through the open wound against the first thin layer of granulation tissue about ten days postoperatively.

In all cases the wound is kept wide open by means of a cotton tampon inserted firmly between its margins; the upper part of the incision in the suprameatal triangle of Lempert must be kept open to allow easy access to the entire cavity during the healing period.

Granulation tissue can thus be kept under control and healing supervised. Once the immediate postoperative reaction has subsided, usually in six or seven days, the dressings need only be changed once or twice a week; postoperative care consists of merely keeping the cavity clean, the meatus open, and of controlling granulations, preferably by removal with the sharp curette should they become exuberant.

CONCLUSIONS

1. There are so many practical advantages of the endaural method of approach for surgery of the temporal bone that it should be called to the attention of all otologists.

2. The method requires a technique that is new to most otologists; it demands new skills and some specialized equipment.

3. The endaural approach and the employment of the modern technique of mastoid surgery developed by Lempert, Shambaugh, Meltzer and others have and will undoubtedly continue to have a large effect in a renaissance of otologic surgery; all credit is due to those who have given us this new means to more perfect surgery of the temporal bone.

140 EAST 54TH STREET.

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XXXV

THE THYROID AND THE NOSE

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For a long time it has been my impression, more or less casual, that certain indolent and intractable nasal conditions were peculiar to the type of individual regarded as hypothyroid. These conditions, usually of such a low order of acuity that they should have responded to simple measures, existed in pale asthenic individuals as a rule but not invariably; only occasionally was there a spontaneous complaint of chilliness or of dry skin and hair.

When a closer study of the records revealed that almost 90 per cent of the suspects sent for a determination of the basal metabolic rate turned up with sizable deficiencies, it seemed worth while to coordinate the data and to present them to this meeting.

The survey comprises 84 cases personally seen in private practice. Of this number 34 patients were known previously to be hypothyroid; 50 were sent first by the laryngologist for basal metabolism tests.

The appearance of the nasal mucosa in these cases was not uniform either in color or texture but there was one dominant characteristic which is difficult to define. It may best be characterized as "indolent and unresponsive" both as to its physiological state and its reaction to treatment. In 75 per cent the membrane was slightly red, inclined to stickiness and even dryness, sometimes a little bloody and of a poor texture. It might be said to be "chapped." In the remaining 25 per cent the membrane was pale and sometimes boggy. Less than half of the latter type was found in demonstrably allergic subjects, although the series is not large enough to make this proportion significant. The change gave the impression of being nutritional rather than vasotonic or essentially infectious.

Presented before the American Laryngological Association, St. Louis, Mo., April 23, 1947.

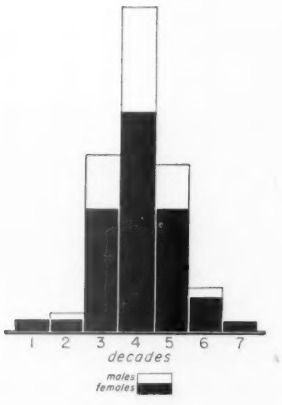

Fig. 1.—Incidence in the various age groups.

In the "chapped" type the average basal metabolic rate was minus 17%; in the pale, boggy type minus 20.8%. It was not surprising to find headache of some description present in 40 of the 84 patients. Asthenia was the (unprompted) complaint in 15, and chilliness in only 4.

Twenty-four of the series were males, 60 were females. In both sexes the fourth age decade predominated overwhelmingly.

Among the chief complaints nasal obstruction appeared 29 times; headache, 40 times; frequently recurring or continuous cold, 30 times; rhinorrhea (as in hay fever), 5 times.

Thyroid extract was administered in all cases, sometimes combined with thiamine chloride, in doses to meet requirements and ranging from 0.5 grains to 5 grains daily. The average was a little over one grain. The results of this treatment were as follows:

Pronounced improvement	37
No improvement	12
Doubtful, incomplete, or possibly improved by some associated measures	35

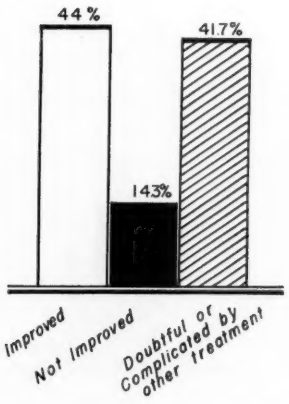

Fig. 2.—Results of thyroid extract administration.

In the group of 50 cases suspected for the first time as a result of nasal examination alone the metabolic rate was:

0 to —10%	9 cases
—11% to —20%	18 cases
—21% to —30%	8 cases
—31% to —40%	4 cases

In 5 cases the rate was increased (plus 3% to plus 15%).
In the 6 remaining cases exact figures were not available.

The series is as yet too small to make statistical details of much value but the facts: 1.) that thyroid deficiency could be suspected by the rhinologist from the appearance of the membrane and its failure to respond to treatment, 2.) that the deficiency could be demonstrated in 39 of 44 suspected cases, and 3.) that pronounced improvement followed administration of thyroid in 29 (compared to 8 not improved, 13 doubtful or incomplete) establish the local symptomatology at least to our own satisfaction.

The foundations upon which the preceding findings rest are generally accepted. To quote from Byrom,[2] the cells in their normal environment are surrounded by a "freely circulating, relatively protein-free ultrafiltrate of the blood plasma, the secretion of which is regulated by the hydrodynamic pressure within the capillaries op-

posed by the colloidal osmotic pressure of the plasma protein." What protein there is, is of two kinds: first, the dispersed protein (about 1%), second, the so-called ground substance, a fixed colloidal gel which acts as a scaffolding or cement to maintain the structural form of the tissue.

Byrom is of the opinion that myxedema consists of an expansion of the ground substance into the circulating substances.

Knowing the highly vascular nature of the nasal tissues and considering the looseness of their integration, it is entirely possible that something resembling a myxedematous process may appear here under conditions too minor to cause general myxedema in more rugged tissues. Certainly the symptoms arising in the nose which respond to the administration of thyroid substance: namely, extravasation, changes in the nature of the surface fluids, permeability, malnutrition, swelling and edema, could reasonably result from the process suggested by Byrom. All of them would render the tissues more susceptible to bacteria and just possibly to antigens.

In this connection may be mentioned an observation by Hirschberg and Bronstein[4] regarding the effect of thyroid deficiency upon agglutination following typhoid inoculation. They found briefly that in a 22-year-old cretin the agglutination test remained persistently negative following typhoid vaccine. Only after the administration of thyroid in one grain doses could partial agglutination be observed, which became strongly positive after the administration of two grains daily for six months.

This is the only report one was able to find relating to human subjects. The literature on animals is confused. Some investigators report an increased, others a decreased immune reaction in thyroidectomized animals.[5] It is significant, however, that all of them report some alteration.

Relations between nephrosis and hypothyroidism have been suspected[1] and other processes which may well be analagous to those noted in the nose have been described in the vestibular apparatus,[7] in the cornea,[6] and in the endometrium.[3]

"Broadly speaking," writes Means, "[the thyroid hormone] has to do with the rate of growth and differentiation of tissues, with the rate of energy exchange, with the metabolism of proteins, carbohydrates, fats and lipoids, salts and water, with the irritability of various tissues and with the activity of other endocrines."

Commenting upon a suggestion of Moehlig[8] that the thyroid hormone has a selective action upon ectodermal tissues, Means[6] (page 48) states as his belief, "that the thyroid hormone acts on all cells, not only on those of ectodermal origin. The effects on ectodermal structures simply happen to be very conspicuous."

In any case there is reason to believe that the nasal tissues are so affected. Sweat glands are known to be influenced by the hormone and I have on more than one occasion called attention to the related activity of these and the glands of the respiratory mucosa.

At the risk of being prematurely didactic I should like to offer the following observations:

1. That deficiency of the thyroid hormone may result in changes in the nasal mucosa which can be recognized as such.

2. That these changes are of two types: a) red, dry, irritated, "chapped" or desquamative; b) pale and boggy.

3. That patients deficient in the thyroid hormone may have an increased tendency to nasal infection and nasal allergy which can be corrected by the administration of thyroid extract alone.

4. That headache and nasal obstruction are prominent symptoms.

5. That the basal metabolic rate is helpful but not infallible in distinguishing cases suitable for treatment with thyroid extract. Five patients of the 37 who showed improvement had rates between minus 10 and minus 3, and one had a rate of plus 3.

6. That the basal metabolic rate is not a reliable prognostic index. Four patients showing the greatest improvement had basal rates of -4, -9, -11 and -34 (later -12), respectively. In all cases the response of the patient was considered more useful than the basal metabolic rate in determining the most effective dosage of thyroid extract.

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XXXVI

SOME PHYSICAL PROPERTIES OF THE CONDUCTION APPARATUS

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Renewed interest in surgery of the conduction mechanism has brought into focus our limited knowledge of the acoustic properties of this part of the ear. The standard reference of auditory function is the threshold curve. This is a composite of the action of all the different parts of the auditory system. The contribution of the conduction apparatus and the physical dimensions that determine this are not entirely clear. The auditory system is not uniformly sensitive. The frequency range and the shape of this sensitivity curve depend in part at least on the parts of the ear that interact physically with the stimulus. The properties of any matter that determine how it will respond to a vibratomotor force like the sound wave are mass, elasticity, and resistance or damping. In general, sound stimuli in air have so little energy that a small mass is needed to react with it. Elasticity is needed to restore the mass to its original position. The amount of resistance or damping determines how soon and in what manner the mass will return to rest after being displaced. The amount of mass and elasticity will determine the resonance frequency of the structure and this is proportional to the square root of elasticity divided by the mass. A stimulus of this resonant frequency will produce the greatest amplitude in the system. A highly resonant recording system has the disadvantage of being extremely sensitive over a small range. This can be overcome in two ways: by increasing the resonance frequency far beyond the range you wish to record, and by increasing the damping or resistance. The acoustic physicist can define the physical constants and construct a receiver to react in a given way. The otologist, on the other hand, is dealing with a given receiver and must analyze its physical properties in order to know how it will react to the sound stimulus.

Division of Otolaryngology, The University of Chicago.

Delivered in part before the American Otological Society, St. Louis, Mo., April 21, 1947.

This work was aided by grants from the Douglas Smith Foundation for Medical Research of the University of Chicago.

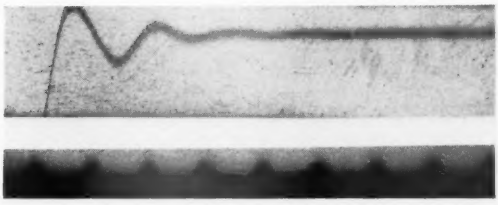

Fig. 1.—Movement of the head of the malleus with intact chain when suddenly released from a displaced position. Timing wave below 1000 cycles per second.

The determination of some of the physical properties of the conduction apparatus can best be done on fresh cadaver ears. Bekesy[1] has pointed out that the elastic properties of the conduction apparatus in fresh specimens are similar to those in the living. It is possible to preserve the elasticity of such specimens for some weeks if they are kept in a cold, moist chamber, using 1/4000 aqueous merthiolate in physiologic sodium chloride solution. The oscillations of the ear can be induced by sound stimuli delivered through tubing to the external canal or the middle ear. The sounds are generated by a loud-speaker driving unit activated by a suitable audio-oscillator and amplifier. Oscillations can also be induced by single large pulses, such as the shock pulse of an explosion or an electric spark. The oscillation is observed by mounting small mirrors on the part to be studied. With an arc light this becomes part of an optical system that amplifies the movement and recording of the oscillation may be made on moving film with a suitable camera.

Another method that we have used was developed by Kalmus[2, 3] and is based on amplitude modulation produced by movement of a small piece of metal foil near a tuned circuit. The part of the acoustically driven conduction apparatus moves a small metal foil placed upon it. The foil is set in motion in close vicinity to the inductance of the resonant circuit in a radio-frequency oscillator. This relative motion varies the mutual inductance between foil and coil, thereby changing the resistance reflected into the coil. This action produces amplitude modulation of the oscillator by varying the loss of the resonant circuit. The amplitude modulation is detected in such a way that small gap changes have no influence on the output. Furthermore, the mechanism as a whole can be displaced

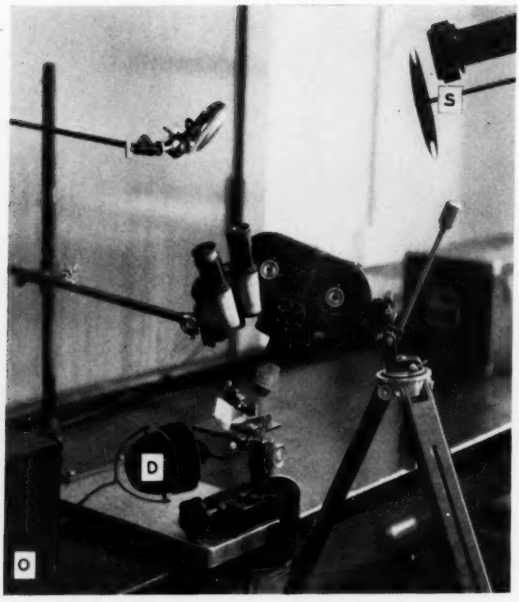

Fig. 2.—Arrangement for recording acoustic oscillation in the fresh specimen with a camera. D, driver unit; O, audio-oscillator; S, stroboscope.

from its resting position with a small hook around the incus and the manner of oscillation, after the hook is suddenly released, will be determined by the inherent properties of the coupled chain.

These oscillations can be recorded.[4] If you displace the conducting mechanism away from center in this way and record the movement of the head of the malleus, you see that, on suddenly releasing the hook, the oscillation will be very brief and have a certain frequency. This frequency is determined by the mass and elastic factors of the conduction apparatus as a whole when coupled to the labyrinth and represents the resonant frequency. The speed with which the oscillations stop is a measure of the resistance or damping of the chain. We see that the mechanism has an over-all resonant frequency of about 750 as recorded at the head of the malleus and that it is highly damped (Fig. 1). The degree of damping is not, however, great enough to prevent overrecording at the resonant frequency. It is, however, great enough to indicate that the mechanism can

rapidly come to rest after the stimulus has stopped and thus prevent aftersensations and insure that the mechanism will be ready to respond to the next stimulus. The fact that the resonance frequency of the conduction apparatus is below that of maximum sensitivity of the ear as expressed in the threshold curve is interesting. The ear is most sensitive at about 3000 cycles. This would suggest that some physical properties of the cochlea itself may be operating, such as the windows, the fluid or perhaps the leverage action of the basilar membrane with respect to the outer hair cells, or that this sensitivity curve is due to some property of the nervous system itself.

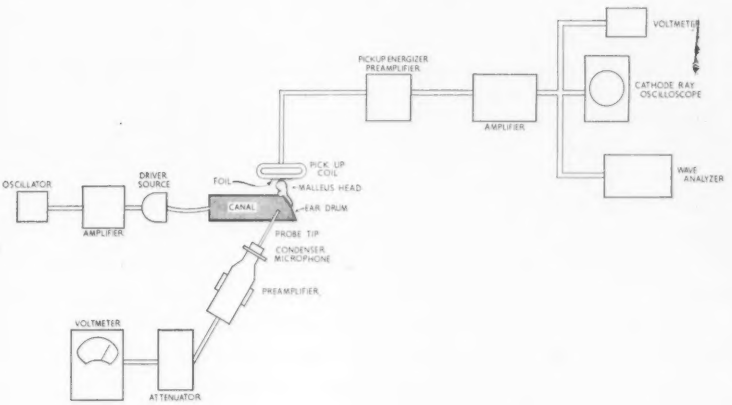

Fig. 3.—Diagram of the experimental setup for studying acoustic oscillations of the conduction apparatus with the magnetic flux method.

Bekesy,[5] using a "nul" method for measuring volume displacement of the round window in the fresh cadaver ear, found that the amplitude of vibration of the round window in response to a constant sound pressure at the drum is almost independent of frequency from about 150 to 3000 cycles. Bekesy[6] investigated many aspects of the physics of the conducting mechanism and the labyrinth including the round and oval windows. By studying the phase shift between the delivered sound in the external canal and the movement at the round window he could determine through what frequency range the vibrating system was stiffness-controlled or mass-controlled. The sound frequency that resulted in the zero phase shift represented the resonance frequency of the vibrating system. According to known laws of mechanical impedance of vibrating systems, a

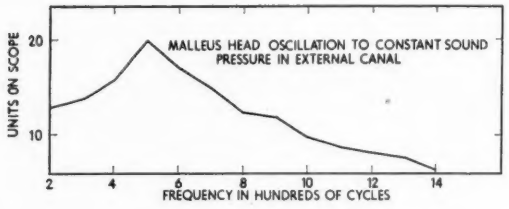

Fig. 4.—Resonance frequency and damping of the chain as recorded at the head of the malleus.

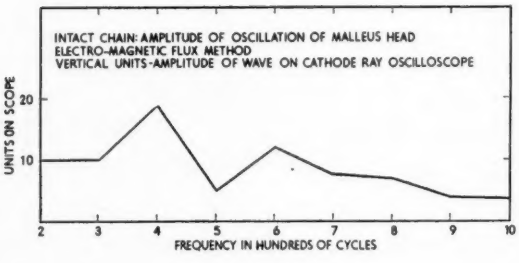

Fig. 5.—Resonance frequency and damping of the chain as recorded at the head of the malleus.

stiffness reactance produces an advance of the phase of oscillation over the phase of the stimulus. At resonance no phase shift is observed and beyond resonance the system is controlled by mass reactance and the phase of the induced vibration lags behind the phase of the stimulus. With both windows removed the cochlear fluid vibrated as a mass-controlled system. With the round window intact and the oval window removed, the vibrations were stiffness-controlled up to 1400 cycles, which was the resonant frequency. With both stapes and round window intact the cochlear fluid vibrated as a stiffness-controlled system throughout the frequency range tested—up to 3000 cycles. When the entire conduction system with the drum and the inner ear was stimulated, the vibrations resembled that of an elasticity-controlled system in the lower frequencies. At higher frequencies the effect of friction becomes more important.

The acoustic oscillations of the component parts of the conducting mechanism can be seen in the living and in fresh cadaver ears, if the sound intensity is sufficiently great and the movements are observed with stroboscopic light. This was well known and extensively reported on by Mach and Kessel[7] about 70 years ago in the living and in fresh specimens. Instead of using organ pipes and sunlight interrupted by a tuning fork as they did, one can more conveniently use a loud-speaker driving unit and a mechanical stroboscope with artificial light (Fig. 2). In the living the magnification of the Siegle speculum is sufficient. In the cadaver preparations, greater magnification as from a microscope or that obtained with moving picture projection can be used. Direct observations of the submolecular displacements at threshold would be ideal but have not been possible. Low frequency sounds are best used in the living and in fresh preparations since they are well tolerated and it is easy to build a low frequency mechanical stroboscope, one of 180 cycles per second.[8] A stimulus of about 100 decibels above threshold is needed to obtain easily visible movement. In the cadaver preparations a magnification of about 200 is easily obtained at about one meter from the head of the malleus when recorded optically from oscillograph mirrors. Visible displacements over a wide range of frequency can be obtained in these fresh specimens. In general the oscillations are reduced in amplitude as frequency is raised so that at about 1000 cycles very little is seen.

The oscillation induced in the malleus is greater than that in the stapes. Both motions are also asymmetrical. A greater displacement takes place on the negative side of center corresponding to the drum moving outwards. This asymmetry is more marked at the

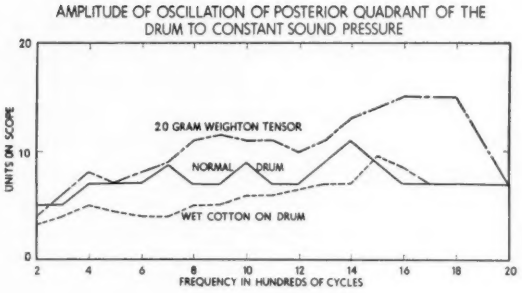

Fig. 6.—Response of the drum to constant sound pressure in the middle ear. Effect of changing mass and stiffness of the drum.

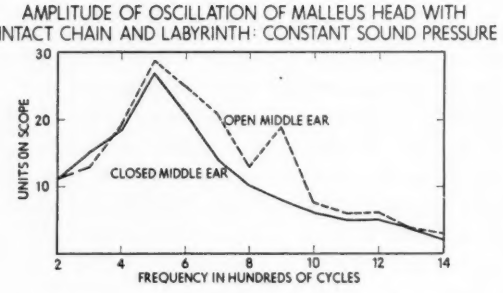

Fig. 7.—Effect of the air sealed in the middle ear on the resonance and damping of the ossicular chain.

stapes. The pull of the tensor muscle has a greater effect on this negative phase of movement and tends to shift the axis in the opposite direction.

With the electromagnetic pickup which we used, the amplitude of oscillation can be observed on the cathode ray oscilloscope (Fig. 3). At the same time a constant sound pressure at various frequencies can be delivered to the preparation through tubing. A calibrated condenser microphone with probe tip is needed as a guide in producing constant pressure. This was made available through the courtesy of the Bell Telephone Laboratories and the cooperation of a member of their technical staff, Mr. Mark B. Gardner.

Recording amplitude of oscillations versus frequency gives us another way of studying the resonance frequency and the damping of the various components of the ear.*

For constant sound pressure there is not uniform amplitude of oscillation at the head of the malleus. The maximum amplitude appears to be at about 500 to 600 cycles (Figs. 4 and 5). The sensitivity of the magnetic flux system which we used did not permit a usable signal above 2000 cycles. The resonant peak is quite broad, suggesting considerable damping. Oscillation of the drum is even more highly damped as suggested by the amplitude versus frequency response (Fig. 6). The small volume of air enclosed in the middle ear contributes to the elasticity and damping of the conduction apparatus (Fig. 7). With the chain separated we can learn something of the reaction of the individual components of the oscillating system. When the drum and its attached malleus alone are set into vibration, we observe maximum amplitude at the lowest frequency (200 cycles) used in the test and a rapid reduction of the amplitude with constant sound pressure (Fig. 8). This suggests a mass effect with a low resonant frequency. When the drum, malleus and incus are set into vibration as a unit and the amplitude recorded at the end of the long process of the incus, a curve more nearly resembling that of the malleus head in an intact chain is obtained.

There are many physical phenomena to be studied in clarifying the interaction of the conducting mechanism and the molecular oscillations called the sound wave. But it is apparent to us as clinicians that as we learn more about these important physical phenomena we can better understand the great variety of threshold curves seen in conduction lesions.[9]

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*These experiments were carried out with the assistance of Mr. J. Hind and Mr. S. Adams.

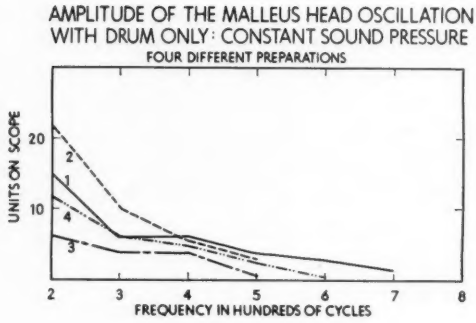

Fig. 8.—Response of malleus with only the drum attached. Four preparations.

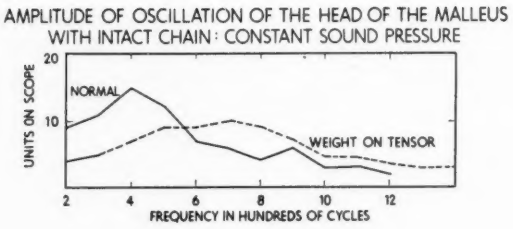

Fig. 9.—Change in resonant frequency and damping of the ossicular chain due to an increase in stiffness.

Bekesy[10] studied the amplitude of vibration of the drum and the stapes footplate for constant sound pressure with a specially devised electrical method using a variable capacity probe. He was able to detect movements as small as 10^{-6} cm. with his apparatus. He found a uniformity of amplitude of drum movement for the frequencies used (between 100 and 2400 cycles). He determined the contours of uniform amplitude over the drum and found that the drum moved as a rigid cone centered at the umbo up to 2400 cycles. Above this frequency the malleus does not faithfully follow the drum movement. The most convex portion of the drum did not show the greatest movement; this is contrary to the widely held opinion that this convexity affords an amplifying factor for forces delivered to the malleus. The amplitude of stapes movement measured at the vestibular side of the footplate versus sounds of constant pressure at the drum was different from that found for the drum. The amplitude of stapes movement decreased progressively with frequency; that is, the mechanical impedance of the conduction apparatus as measured at the footplate was a resistance rather than an elasticity or mass reactance. By a "nul" method Bekesy determined the sound pressure at the footplate as compared with that at the drum and found a rather uniform pressure amplification factor of about 20 for the frequencies used (up to 2400 cycles). He studied the effect of cutting the axis ligaments of the chain on the pressure transfer from drum to stapes and the effects of a static air pressure of 10 cm. of water on the drum on this transfer. In the latter experiment, he observed a marked effect in reducing transfer from drum to stapes for frequencies up to 800 cycles.

It is evident that conduction lesions are only partly described anatomically. The physical changes in the apparatus as an acoustic receptor determine the resultant response to the frequencies presented for threshold tests. These physical changes alter the ability of the ear to absorb the energy of the air-borne sound wave. Menzel[11] and later Metz[12] have demonstrated the feasibility of making objective measurements of some of the acoustic properties of the normal and the pathological ear in the clinic. Adopting the apparatus devised by Schuster, Menzel studied the absorption of sound by the normal and the pathological ear and Metz determined the acoustic impedance of the ear in patients with various conduction and inner ear lesions. These determinations give information about the efficiency of the ear in absorbing the energy of the sound wave in air. It is expressed by the ratio between the intensity of the sound wave reflected from the drum and the intensity of the incident sound wave together with

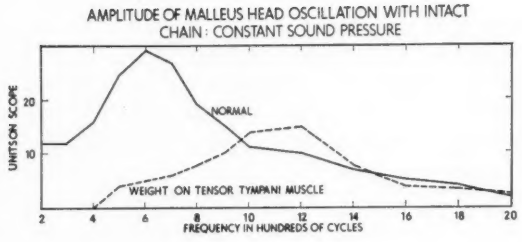

Fig. 10.—Change in resonant frequency and damping of the ossicular chain due to an increase in stiffness.

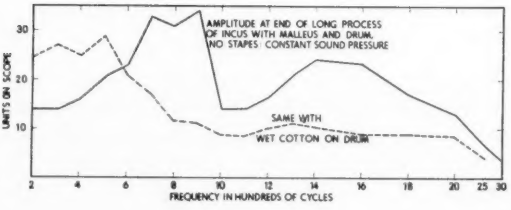

Fig. 11.—Change in resonance and damping of a malleus-incus preparation due to an increase in the effective mass of the drum.

the degree of phase shift taking place at the drum between the pressure of the reflected wave and the pressure of the incident wave. Both Menzel and Metz found a definite decrease in the absorption of sound by the ear in some conduction lesions and Metz observed a similar effect during the reflex acoustic contraction of the middle ear muscles.

From an acoustic standpoint, lesions may be expected to produce preferential changes in sensitivity versus frequency more fully expressed in the term "impedance". Thus an acoustic receiver can be changed by altering its elastic properties or stiffness. In general an increase in the stiffness increases the resonant frequency of the system and reduces the amplitude of response or sensitivity for low frequencies and increases the amplitude at the higher frequencies. Since the oscillations are recorded below the point of maximum sensitivity of the ear, the latter effect is not so prominent in the threshold curve, but low frequency loss can be seen. Acute changes in air pressure on the two sides of the drum are an example of a condition producing a stiffness change. Experimentally, stiffness change in the preparation can also be produced by pulling on the suitably isolated tensor tympani muscle (Figs. 9, 10). As to the effect of changing the mass of a vibrating system, in general the greater the mass, the more difficult it is to move it at the higher frequencies and the lower is the resonant frequency. Changing the mass of the drum by a bulla or granuloma is a clinical example—as is perhaps the presence of fluid in the middle ear, for while the mass of the adherent air molecules near the drum do not greatly affect its oscillations, the adherent water is acoustically important as a mass increasing factor for the drum. High frequency losses in the threshold curve may be explained in this way.

In the patient and in the preparation, the drum can be loaded with wet cotton and the effect on amplitude of oscillations or the threshold curve may be observed (Fig. 11). While the response of a single vibrator like a diaphragm can be predicted by known laws of acoustics that take into account mass, elasticity and damping, it is evident that response of a system like the conduction apparatus is much more difficult to understand since it is made up of several masses connected by a number of elasticities. These are mutually interacting in such a way as to produce a small amplitude with great pressure at the footplate from a relatively large amplitude with small pressure at the drum. This is in the proper direction to give a better match between the acoustic impedance of the fluid in the internal ear and the air in the canal. Acoustic impedance is dependent on the density

of the medium and the velocity of sound in the medium and is about 3500 times greater in fluid than in air. If you remove the elastic, restoring force of the annular ligament, the stapes moves with a much greater amplitude when driven by the intact chain. This elastic factor of the annular ligament interacts on the rest of the elasticities in the conduction apparatus, as at the joints and tendons and ligaments. The coupled masses and the elasticities existing in the conduction apparatus constitute a mechanical leverage system by which the acoustic impedance of fluid is more nearly matched to that of air.

Lesions of the conduction apparatus will produce a variety of threshold curves depending upon the manner in which the masses, elasticities, and resistances of the component parts are affected.

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XXXVII

PRESENT STATUS OF DIPHTHERIA

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The present interest in diphtheria is probably due to fear of spread of a new virulent type for which antitoxin is more or less ineffectual and conditioned by a prior idea that diphtheria had been quelled. Neither of these ideas is entirely true.

Diphtheria has shown a very marked drop in mortality and this has extended over a long period. If we study the curve of mortality in Boston we note a considerable lowering of the death rate shortly after antitoxin was begun in 1895, followed by a stationary and relatively low rate. This represented the level of lay and professional ignorance regarding diphtheria because, during the same period, of over 800 cases developing under observation in the hospital no patient died. In other words, this death rate chiefly represented not patients whom antitoxin could not affect but patients who, due to the mild subjective symptoms and insidious course, were delayed in receiving antitoxin.

Following 1920 there has been a marked lowering of mortality which has reached a very low level. This should be zero if it were not for human frailty.

The nature and the severity of diphtheria have not changed in spite of the low mortality. There is no such continued fall in percentage mortality rate as there is in the general death rate. Cases have largely ceased to occur but the ones that do have all the characteristics and dangers of yore. The promptness of diagnosis and treatment and the age of the patient have a marked effect on mortality but the mortality rate at times shows rises and falls not due to known causes.

The marked variation noted in fatality in the past 20 years in the Boston City Hospital, South Department, is probably due to the small number of patients in each year.

Presented before the American Laryngological Association, St. Louis, Mo., April 23, 1947.

From 1895 to 1920, of 33,513 patients the mortality was 10.36 per cent and from 1921 to 1946, of 5478 patients the death rate was 9.9 per cent, so it is evident that in Boston the nature of diphtheria has not much changed.

The severe diphtheria which occurred in Europe and Great Britain about 1925 seemed to be explained as the new type (gravis) demonstrated by Anderson and others in 1931 at Leeds. This gravis type was distinguished bacteriologically by the colony on tellurite agar and physiological differences in sugar splitting.

TYPE	STARCH	GLYCOGEN	BOUILLON	HEMOLYSIS	ACIDITY	COLONY ON TELLURITE AGAR
Gravis	+	+	Granular Surface growth	O	Above pH. 7.4	"Daisy head" grey-black
Mitis	O	O	Turbidity No surface growth	+	Below pH. 7.3	Shiny black
Inter- medius	O	O	Granular No surface growth	O		Grey-black, slightly crenated

Toxin type and production were not unusual. The mortality rate in Leeds (gravis 15 per cent, intermedius 7.8 per cent, mitis 0.0 per cent) was confirmed by many workers.

Robinson and Marshall, in Manchester, reported the mortality rate from the gravis type to be 15.1 per cent; from the intermedius type 8.7 per cent; from the mitis type 0.5 per cent. The explanation for the increased virulence and the peculiarity of behavior was various and not always sound or confirmed by others. Pappenheimer and Johnson had shown, in 1936, that the concentration of iron had a marked effect on toxin production, 0.08 mg. per ml. being the optimum concentration with very narrow limits beyond which toxin production dropped extraordinarily. Mueller showed, in 1941, some strains of gravis much less affected by iron concentrations that had a marked reducing effect on regular strains. No explanation of the failure of antitoxin to affect the disease or of antitoxic protection was made that seemed satisfactory.

In 1941, an outbreak due to the gravis strain occurred in Halifax and was reported by Wheeler and Morton. However, the mortality was only 3.69 per cent. Even if this low level were due to the in-

creased age incidence from the influx of young adults during war time, it would be well within our usual mortality, even if doubled.

In the Baltimore epidemic of 1941, Frobisher found no gravis strains although the "bull neck" type was common and the mortality was 15.1 per cent. He found a strain of C. diphtheria, characterized by very small tellurite colonies and slight but variable splitting of glucose, which he called the minimus. Frobisher has found many nonvirulent gravis strains in the United States.

In the epidemic of the gravis type in Somerville and Boston last year, the death rate was not unusual. Our mortality in 1946 was 6.3 per cent.

One must conclude that, although fluctuation of virulence of diphtheria occurs, it is not associated with the development or introduction of a peculiar, fixed strain. The apparent ineffectuality of antitoxin is more likely to be related to the time of treatment and the speed of toxin action. If antitoxin is delayed more than 24 hours after the onset of the infection, some deaths occur and the percentage death rate rises rapidly with each day's additional delay. In spite of a large and adequate dose of antitoxin, inflammatory action may continue for from 36 to 48 hours. Without doubt, this is due to toxin fixed before antitoxin was given and to the natural time of reaction after toxin is fixed. Even with very virulent cases and a high mortality, contact with others has not resulted in any deaths in over 800 cases seen by the working staff of this hospital before 1920, so that it does not seem impossible that all cases, *if seen and treated at once* at the onset of any symptoms, will respond favorably to antitoxin. This importance of time in antitoxin administration is the reason that generations of medical students have been taught to give antitoxin at once to all patients with mucous membrane infection in whom diphtheria cannot be certainly excluded.

The conception of antitoxic immunity to diphtheria in Schick-negative persons has often been too dogmatic. Immunity is relative and it is impossible to set the level of immunity that will protect under all conceivable conditions of infection and virulence. There have been many instances of Schick-negative persons having diphtheria, although the cases admitted to the hospital in Boston occur chiefly (90 per cent) in unimmunized persons. In the Baltimore epidemic 63 per cent had prior immunity. Ipsen reported, in 1946, two fatal cases in which the blood antitoxin level before treatment was 100 and 32 A.V. per ml. respectively. In our cases at the hospital

it is very rare to find high antitoxin levels. I do not know the explanation of these unusual occurrences.

In Ipsen's cases, all previously immunized patients recovered, although deaths occurred in persons with spontaneous levels above 0.01 u. per ml.

Immunization with two doses of alum-precipitated toxoid or aluminum hydrate absorbed toxoid, at intervals of three weeks to three months, or three doses of soluble toxoid every three to six weeks, is satisfactory. Booster or recall doses of toxoid every three to five years are considered desirable although not yet proved essential. The response to the booster dose is rapid (four to seven days) but a little too slow for the safety of a person after infection has occurred.

All children should be given toxoid or a similar antigen, preferably between the sixth and eighth months of age.

For treatment, despeciated antitoxin treated chemically and with pepsin-HCl or a similar digestant has a great advantage, being low in bulk and markedly free from protein reactions as compared to the high percentage (80) of reactions from crude antitoxin such as was produced by Theobald Smith. Reactions usually occur in less than five per cent and severe reactions are extremely rare.

The addition of antibiotics, such as sulfonamides, and penicillin, have had no effect on the treatment of diphtheria but are of extreme value in reducing the dangers from secondary infections, especially from the pneumococcus and the streptococcus. Penicillin adds nothing of benefit to the treatment of diphtheria, which should always be treated with antitoxin if any treatment is required. But penicillin has proved to be the best treatment available for the carrier state. Carriers are almost invariably rendered bacteria-free in from four to seven days with large intramuscular doses—about 200,000 units per day for one week. We have not been able to secure prolonged freedom from bacteria with local penicillin.

Weinstein has recently reported excellent results in carriers.

One of the urgent problems resulting from diphtheria is obstruction of the airway. While this may occur at any point from the throat downward, it is most common at the larynx. For some years opportunity for medical students to learn methods of relief have been reduced to a point near zero. The tendency to revert to tracheotomy instead of intubation may be due to this fact. We have no comparative figures for intubation and tracheotomy, although we still believe that intubation is preferable. The necessity of special

techniques and instruments, as well as special operative supervision by trained personnel, for intubation is probably slightly greater than for tracheotomy. We use, chiefly, laryngoscopic intubation as first suggested by Mosher. The chief danger in intubation has been the development of bronchopneumonia and this has been largely eliminated by antibiotics. This is also true in tracheotomy.

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XXXVIII

"MÉNIÈRE'S"—A SYNONYM OF CONFUSION

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In 1848, Ménière,[1] in his translation of Kramer's Treatise on Diseases of the Ear, first reported his case of "A young girl stricken with complete and absolute deafness in a few hours. Death, which came a few days after, gave me the opportunity to dissect carefully the two temporal bones and I found throughout the labyrinth a plastic, reddish lymph, which appeared to be the result of an exudation from all the membranous surface lining the inner ear."

In 1861, he read a paper before The Academy of Medicine in Paris, in which he again reported the same case.[2] He stated:

> "I have spoken elsewhere, a long time ago, of a girl who, having journeyed by night, in winter, on the box seat of a stagecoach during the time of her catamenia, became, as a result of the severe cold, completely and suddenly deaf. Admitted to the service of M. Chomel, she presented as the principal symptom continuous vertigo—the least effort at movement caused vomiting, and death took place on the fifth day. The autopsy showed that the brain, the cerebellum and the spinal cord were absolutely free from any change, but, as the patient had become completely deaf after having always heard perfectly, I removed the temporal bones in order to investigate thoroughly what could be the cause of this complete deafness which had come on so rapidly. The only lesion I could find was in the semicircular canals, which were filled with a red, plastic matter, a sort of bloody exudate, a few traces of which were discovered with difficulty in the vestibule and which did not exist at all in the cochlea. The most careful investigation allowed me to establish with all desirable precision that the semicircular canals were the only parts of the labyrinth which showed an abnormal state, and this consisted, as I have said, in the presence of a reddish, plastic lymph, replacing the liquid of Contugno."

Thus confusion regarding this condition starts at the fountainhead. In one report he gives sudden deafness as the outstanding feature and, in the other, continuous severe vertigo.

Pathologically, in one report he states that the exudate was found throughout the labyrinth and in the other that it was limited to the semicircular canals. Unfortunately, no previous history of the case is given, nor does Ménière state the cause of death, which

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Remarks by the President at the 51st Annual Meeting of the American Laryngological, Rhinological and Otological Society, St. Louis, Mo., April 25, 1947.

could not have been due to meningitis, however, because the brain and meninges were perfectly normal, nor does he suggest in either report that it was a labyrinthine hemorrhage.

His original description of the symptomatology of this condition[3] is a classic and even now is superior to that of many subsequent writers. He states:

"An auditory apparatus, perfectly sound, can become all at once the seat of functional disturbances, consisting of noises of various character, continuous or intermittent, and these are accompanied by more or less great diminution of hearing. These functional disturbances having their seat in the internal ear can often cause attacks considered cerebral, such as vertigo, staggering, uncertain gait, rotation and falling, and accompanied besides by nausea, vomiting and a syncopal state. The attacks which are of intermittent form are soon followed by deafness, more and more grave, and often the hearing is even suddenly and completely obliterated. All this leads us to believe that the material lesion, which is the cause of these functional disturbances, resides in the semicircular canals."

Ménière receives and deserves unbounded credit for demonstrating a labyrinthine lesion to account for these symptoms, and his place in medical history is secure.

However, one of the chief factors in the average individual's difficulty in evaluating the symptom of vertigo is the indiscriminate use of Ménière's name in connection with cases of vestibular disturbance due to various and sundry causes, which do not correspond, either clinically or pathologically, with Ménière's original description.

Each author gives his own definition of "Ménière's disease," which term he often uses interchangeably with "Ménière's syndrome" or "Ménière's symptom complex."

A cursory scanning of recent otological literature reveals many instances illustrating the confusion resulting from this indiscriminate use of Ménière's name.

Author A states: "Ménière's disease is not a disease sui generis but a typical reaction of a predisposed labyrinth to an almost infinite series of exo- and endogenic influences, which, however, have this in common, that they express themselves through the vessels, especially the capillaries."

Author B says: "Ménière's disease or perhaps more correctly Ménière's syndrome is characterized by a sharply defined group of signs and symptoms that is almost pathognomonic: Sudden and recurring attacks of dizziness, nausea, vomiting, partial deafness and tinnitus. The two essential features of the syndrome are dizzy spells and deafness. Ménière's disease may be unilateral or bilateral. Pseudo-Ménière's disease is Ménière's disease minus the deafness and tinnitus. When dizzy attacks precede unilateral deafness and tinnitus, the diagnosis of

pseudo-Ménière's disease must be made. If a patient has recurring attacks of dizziness, the diagnosis is either Ménière's disease or pseudo-Ménière's disease, other signs or symptoms notwithstanding. That there is an organic lesion is, of course, evident at once from the omnipresent deafness. . . . Since both the vestibular and cochlear divisions of this nerve are involved, it is evident that the lesion is in the nerve itself and not in the end organ."

"Ménière's disease has probably nothing to do with the inner ear. It is merely an explosion of the functions of the inner ear and is due to a lesion of the auditory nerve.

"We know of no cause of Ménière's disease. Certainly there is no infection. Never does Ménière's disease follow infections of the middle ear. And so far as we have been able to learn from our series of cases there has been no focal or systemic infection to which this condition might be ascribed. It is necessary to assume that we are dealing with a lesion not in the middle ear nor in the inner ear, but rather in the auditory nerve. A lesion of the inner ear could not affect both the acoustic and auditory (vestibular?) divisions of the eighth nerve."

Author C—States that a disturbance of the chemical or nervous control of the auditory artery causes Ménière's disease.

Author D—States that in Ménière's syndrome there may be either a primary vasodilator or a vasoconstrictor mechanism at work. Later in the same article he speaks of the treatment of patients with "Ménière's disease" of the vasoconstrictor type.

Author E—States that microscopic studies of the temporal bone in cases of Ménière's syndrome show a dilatation of the endolymphatic system, especially in the cochlea and saccule and abnormalities around the saccus and ductus endolymphaticus where the absorption of endolymph is believed to occur. The eighth nerve showed no abnormalities. He further states that it seems justifiable to regard this work as establishing the morphological basis of this disorder.

Author F—States, "The term 'Ménière's disease' as used in this report is not synonymous with 'Ménière's symptom complex' and 'Ménière's syndrome' as they have commonly been used in the past. The term 'Ménière's disease' is herein restricted to this [hydrops of the labyrinth] entity."

Author G—Believes the condition is due to bacterial intoxication of the labyrinth and describes it as "focal labyrinthitis."

Author H—Reported as Ménière's disease a series of cases of epidemic meningitis and two cases of leukemia with sudden deafness and dizziness and one case of tabes with sudden deafness.

Author I—Suggests that a localized central lesion is responsible for Ménière's symptom complex.

Author J—States that, at present, Ménière's disease is used for any collection of aural symptoms for which the user does not know some other name.

Author K—States, "If the attack of vertigo is characteristically sudden and severe and if it disappears, only to recur again, I believe the condition should be referred to as 'Ménière's syndrome' even in the absence of tinnitus and deafness."

Author L—Suggests that Ménière's disease is a local labyrinthine process, most likely an altered permeability of the capillaries, resulting in local edema. He does not believe loss of hearing is necessary for the diagnosis.

Author M—Ascribes the condition to a disorder of normal conditions of pressure and chemical constitution of the endolymph.

Author N—Says, "We are justified in assuming that there is no primary cause of Ménière's syndrome."

Author O—In an article on hydrops of the labyrinth uses the term "Ménière's disease" for this condition simply as a tribute to Ménière, although the pathological condition is quite different.

And so on ad infinitum.

If such confusion prevails among the writers on this subject, what must be the state of mind of those for whom I plead—the readers?

Would it not be better to recognize the fact that the labyrinths are exceedingly delicate organs of equilibrium which can be disturbed by many intrinsic and extrinsic factors and that this disturbance results in the disabling symptoms mentioned. Long lists of conditions associated with vertigo have been compiled, and many of them proved to be etiological factors— at least, to the extent that their removal has been followed by relief.

As commonly used, the terms, "Ménière's disease," "Ménière's syndrome" or "Ménière's symptom complex" do not constitute a diagnosis, as many seem to think, but are merely high-sounding phrases denoting labyrinthine disturbance, the cause of which may be almost anywhere in the body, which cause must be searched for and found before intelligent treatment can be instituted.

How much clearer is the picture conveyed to the mind, for example, by the use of the term "hydrops of the labyrinth" to designate a certain type of these cases.

I was reassured in my feeling in this matter by the following quotation from Politzer:[4]

"The term Ménière's disease, which was originally applied to the apoplectic form of sudden deafness, was later used in a broader sense, and was applied to various diseases of the ear and central nervous system running their course with attacks of dizziness. This fact, therefore, brought about a certain confusion so that now we are very often satisfied with the diagnosis 'Ménière's disease,' 'Ménière's dizziness' and 'Ménière's symptoms' without taking into account the anatomical seat of the affection giving rise to the combination of symptoms."

Finally, when I consulted the Quarterly Cumulative Index under the title "Ménière's disease," I found no references but only the significant words "See Aural Vertigo."

CARLETON BUILDING.

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XXXIX

A STUDY OF THE EUSTACHIAN TUBES AND THEIR ORIFICES AND LUMENS IN PATIENTS WITH LARGE OPERATIVE DEFECTS GIVING DIRECT VISUALIZATION

FURTHER STUDIES OF THE MUSCLES OF PHONATION AND DEGLUTITION

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AND

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The usual textbook description of the eustachian tube is as follows: The eustachian tube is composed of an osseous and a cartilaginous portion, being narrowest near the junction of the two. The osseous portion opens into the middle ear in the upper half of the anterior wall of the tympanum. The cartilaginous portion is pharyngeal and forms two thirds of the entire length. While the osseous portion is always open, the trumpet-shaped pharyngeal end is closed except during the act of swallowing or yawning, when its lumen is temporarily widened by the contraction of the levator and tensor veli palatini muscles, mainly the latter. Air pressure equalization is effected through intermittent openings of the muscular portion of the tube and takes place simultaneously with the act of swallowing.

Schwarzbart[1] described the osseous portion of the tube as being an anterior evagination of the tympanic cavity and always open, as are the epitympanum or the hypotympanum; he gives this bony space the name protympanum. He states that according to Fraser the cartilaginous tube originates from the first branchial cleft, while the protympanum or osseous portion originates along with the tympanic cavity from the second branchial cleft.

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Presented before the American Laryngological Association, St. Louis, Mo., April 23, 1947.

Cartilage, wherever found in the body, serves certain functions: to give strength, contour, and patency, as for example in the larynx, the external ear, the trachea and the nose.

The tubal cartilage is in the form of an elongated triangular sheet with its base directed anteromedially and with its apex at the isthmus where it joins the osseous portion. The cartilage is thickest at its inferior end, narrows gradually superiorly and finally becomes a hook of cartilage which bounds the eustachian canal superiorly and anteriorly, generally in the shape of a shepherd's crook. Two parts are distinguished in the cartilage of the tube, a medial lamina which protrudes posteriorly, and a lateral lamina which is situated superiorly and anteriorly. In a series of cross sections the cartilage near the pharyngeal ostium is in the shape of a right angle, while that of the tube farther away is crook-shaped. A varying number of accessory cartilages are described.

Being of the structure and form to insure patency, why then should the tubal cartilages depart from the usual function and allow the tube to be closed, as described by many writers?

Rich,[2] in his excellent paper on the physiology of the eustachian tube and its related muscles, reviewed the controversial history and after many experiments came to the following conclusions:

1. Normally, the eustachian tubes are closed. They are opened during the swallowing, yawning and sneezing reflexes. They exhibit no independent reflex dilations either periodic or irregular. Normally, they are not opened by respiratory movements, either quiet or forced, and are unaffected by mouth-breathing or by simple elevation of the soft palate (produced by contraction of the levatores palati alone).

2. Although a most important function of the eustachian tube is that of equalizing the atmospheric pressure on both sides of the tympanic membrane, the mere existence of a disturbed pressure equilibrium will bring about no regulatory reflex. Deglutition, as a means of restoring pressure equilibrium to the ear drum, is performed, when necessary, consciously or through habit; but this reflex is never set in motion directly by the stimuli arising from tension of the tympanic membrane.

3. The levator palati, the palatopharyngeus, the internal pterygoid and the superior constrictor muscle of the pharynx (each of which has been variously described as a dilator or constrictor muscle of the eustachian tube) were found to exert, by their contractions, no influence whatever upon the patency of the orifice or lumen of the tube.

4. The tensor palati is the only muscle which is functionally related to the eustachian tube. Contraction of this muscle is always accompanied by a dilatation of the tubal orifice and lumen. There exists no constrictor muscle of the tube. Relaxation of the tensor palati is followed by a passive return of the tubal walls to the condition of approximation which they normally occupy when at rest.

Graves and Edwards[3] describe the tubal cartilages and sections of the tube at different levels. They describe the lumen of the tube from the pharyngeal opening to the isthmus without reference to its being collapsed or closed, saying, "At the isthmus the lumen is still somewhat slit-like, but the vertical height is reduced."

Perlman[4] discussed the clinical entity of the abnormally patent eustachian tube and cites a number of cases. He states that the eustachian tube is normally closed and its walls are held in apposition by the tonus of the tubal muscles.

Shambaugh[5] states, "The normal eustachian tube lies closed in its cartilaginous portion forming a vertical slit. This closure is maintained by a thick pad of fat which surrounds the lateral wall of the cartilaginous tube. During yawning or swallowing the tube opens momentarily, as a result of the action of two muscles, the levator palati and the tensor palati."

Joachim[6] was fortunate in having a patient in whom there was a defect enabling him to visualize directly the eustachian tube and the palate from above and externally. He describes the movements of the soft palate and the labia of the eustachian tube during phonation, deglutition, coughing, retching and yawning, and concludes, "From the observations made on this patient I came to the conclusion that the tube opens during the act of swallowing, as it was possible to look deeply into the gaping ostium, when the patient was examined for a length of time, during which the mucous membrane would become dry. In the position of rest the lips of the orifice of the tube are in contact in their whole extent, and the cleft is turned forward."

Toynbee[7] says the eustachian tube is closed except during muscular action and that the tympanum forms a cavity distinct and isolated from the outer air.

Cutter[8] says the eustachian tube is open normally but closes in deglutition.

Von Troltsch[9] says the tube is normally closed but must open during deglutition.

Semeleder[10] says the tube narrows in all movements.

Patel and Pellanda[11] say that through an opening resulting from the removal of a neoplasm there were seen vague movements of opening and closing of the tube during respiration.

Caldera[12] says the tube is always open.

Cleland[13] states that the tube is always open and momentarily closes in swallowing. He had a patient in whom an ulcerative defect was so situated that the orifice of the eustachian tube could be clearly visualized. He states that the patient's hearing was unimpaired and he heard as well in one ear as the other. He says that the walls of the tube were ascertained not to be in contact, at least for a considerable distance back from the orifice. He says that a stopper placed up into the tube for more than one-half an inch made no observable difference in hearing through the external auditory meatus.

"To disprove the theory that the eustachian tube is usually closed, and is opened in swallowing, we have yet to seek an explanation of the phenomena on which the theory is based, and particularly of the circumstance that when air is blown into the tympanum, displacement of the membrana tympani continues until the act of swallowing is performed. The explanation that a film of moisture occludes the tube, an explanation which must have forced itself on every uninitiated person suffering from coryza, is, I believe, the right one. The propulsion of air through the nares acts powerfully in setting the moisture of the mucous membrane in motion; and in the eustachian tube it sends it back in the contrary direction to that in which it is normally carried by the cilia of the epithelial lining. The transverse diameter of the tube is extremely small, and a very slight accumulation of moisture in any part of it will cause, by capillary attraction, an obstruction sufficiently strong to resist a considerable pressure of air, or possibly may even cause its sides to adhere with the action of a sucker. The sudden closure and alteration of the form of the tube in swallowing, and the equally sudden recovery of its natural form is precisely the operation most likely to burst such an obstruction, expel superabundant moisture, and allow the escape of pent-up air. The disagreeable sensation in the ears in descending in a diving-bell seems to depend on more than one cause, but engorgement of the mucous lining of the eustachian tube and atmospheric pressure at its pharyngeal extremity are things very likely to occasion obstructions of the lumen. Once such obstruction is formed and the freedom of communication of the tympanic cavity with the external air is checked, discomfort may be expected to begin immediately.

"A point in favor of this view, is to be found in the circumstance that when the sensation of pressure on the tympanum has been accidentally produced by blowing the nose or in some other way, if it be allowed to remain, or if swallowing is ineffectual to remove it, it will often, after a little, suddenly disappear with a slight noise when no act of swallowing is being performed."

* * *

Our observations have been made upon five patients in whom there were large operative defects, making possible direct visualization of the palate, the nasopharynx and the regions of the eustachian tubes.

The first patient was a man in whom a tumor mass had been present for five years with marked external deformity for two or three years and almost complete obstruction of the nose. The patient's face was greatly deformed by the presence of a bulging mass

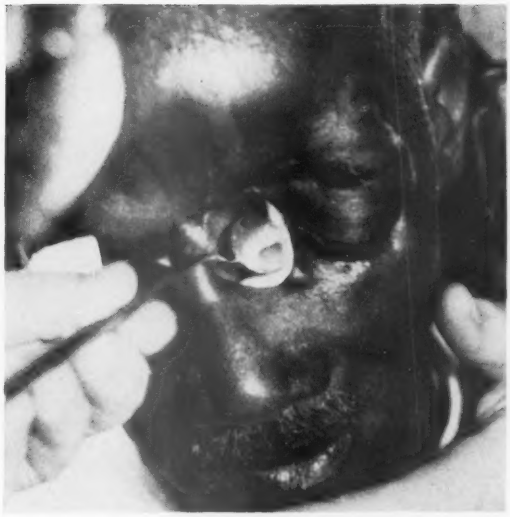

Fig. 1, Case 1.—The open eustachian tube as visualized in the mirror.

situated between the eyes, and extending up to the forehead. The left eye was displaced very much laterally and downwards. The mass was firm and immobile and did not seem to be attached to the skin.

On January 30, 1943, the tumor mass was removed in toto. It was attached to the dura in the region of the posterior ethmoids by a pedicle from its posterior superior aspect. The dura overlying the left frontal lobe was removed with the tumor over an area of about two inches in diameter. There was a true retrobulbar abscess which was drained. All of the sinuses on both sides of the nose were in a suppurative condition. Sulfanilamide was dusted into the wound after hemostasis was secured and the cavity was packed with iodoform gauze. Within a few weeks after the primary operation, all of the sinuses on both sides were exenterated, leaving a large external opening through the bridge of the nose and the sinus region.

The second patient was a colored male, 55 years of age, who had an ulcerative, soft, intranasal tumor which had been present for eight months, increasing rapidly in size. The biopsy was returned with the diagnosis of lymphosarcoma.

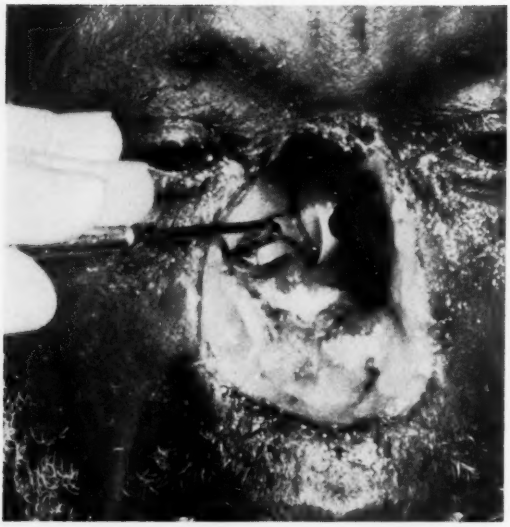

Fig. 2, Case 2.—A combined view of the open eustachian tube: one direct and the other in the mirror.

Under general anesthesia a modified Moure incision was made and the interior of the nose exposed. The tumor was firmly attached to the skin and involved the antrum and ethmoids on the right. The incision was then extended and the entire nose removed along with the involved antrum and ethmoids on the right side. The resulting defect gave an excellent opportunity for study of the nasopharyngeal region.

The third patient was a 63-year-old colored female, who had a large ulcerative lesion of the left eye, orbit and bridge of the nose. The lesion had been present for two years, during which time she had received extensive radiation without beneficial results. A biopsy was done confirming the diagnosis of basal cell carcinoma. The tumor mass was removed in block, with exenteration of the orbit and all of the involved sinuses on this side, leaving a large defect through which the nasopharynx could be directly visualized and studied.

The fourth patient was a 67-year-old colored male who had a tumor of the right malar, alveolar and orbital regions for about

Fig. 3, Case 4.—Eustachian tube as seen directly with a calibrated probe 12 mm. within its lumen.

one year with an obstructing, bleeding mass on the right lateral wall of the nose. A specimen taken from the lateral wall of the nose was reported as squamous cell carcinoma. Under intratracheal anesthesia a modified Moure incision was made. Resection of the right maxilla and all soft tissues except the skin anterior to the prominent portion of the maxilla, and the hard palate as far as the median line, as well as exenteration of the right orbit and all sinuses on the right, was done. The patient made a rather rapid recovery and through the defect in the mouth as well as the orbit an excellent study of the nasopharyngeal region and the eustachian tube was made.

The fifth patient was a 65-year-old white female who had a large adenocarcinoma of the upper jaw, which filled the antrum and destroyed the lateral wall of the nose and the floor of the orbit. Under local anesthesia the tumor mass was removed with exenteration of the orbit and the lateral wall of the nose. The patient responded nicely and at the present time has a large defect through which the nasopharynx and the eustachian tubes can be visualized and studied directly.

Study of these cases demonstrates the following: The movements of the soft palate, uvula, eustachian tube and lateral walls of

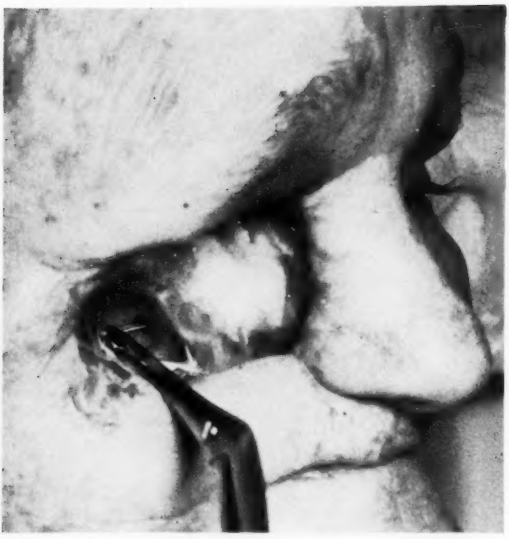

Fig. 4, Case 5.—Direct visualization of the open eustachian tube.

the pharynx in pronouncing the vowels and consonants are the same except for degree.

As the letters are pronounced, the soft palate and the uvula are drawn upward. The lateral walls of the pharynx are drawn upward, backward and toward the midline. There is a slight displacement of the torus tubarius upward and backward and of the region of the mouth of the eustachian tube especially downward and backward, due to the above described actions; but there is no tendency for the tubes to close. If there is any appreciable change, it is that the lumens become larger.

When the patients yawn, the movements of the soft palate, the lateral walls of the pharynx and the lumens of the tubes are the same as in pronouncing the vowels, except that the action is prolonged.

In swallowing, there is no change in the lumen of the eustachian tube in the first four cases, but in the fifth case there is a slight widening of the orifice and the lumen.

In swallowing, the soft palate and the uvula have two movements; the first, a very minor movement, consists of slight elevation of the soft palate in the region of the uvula, with the lateral walls of the pharynx below the eustachian tube being drawn towards the median line. This primary movement is followed immediately by the second, the major movement, which consists of the posterior one third of the soft palate and the uvula being drawn about one-half inch higher than the resting plane. The remainder of the soft palate is drawn downward, the lowest portion being about one-half inch posterior to the attachment of the soft and hard palates. The depression is across the whole extent of the soft palate but is most marked at the middle portion. Following the initial depression of the soft palate and the uvula in swallowing, they are drawn upward, backward and toward the midline along with the lateral walls of the pharynx.

The highest point of the soft palate in phonation or deglutition is 3 - 5 mm. below the level of the hard palate.

There is hermetic closure of the nasopharynx from the hypopharynx during deglutition.

A moving picture, "The Action of the Muscles of Phonation and Deglutition as Viewed Through Operative Wound of the Frontal and Ethmoid Sinuses", was shown at the annual meeting of the American Academy of Ophthalmology and Otolaryngology in 1944.

The anterior labium of the eustachian tube (the salpingopalatinus muscle) extends anteriorly and inferiorly in the superior part and then runs vertically downward to the soft palate, disappearing as it joins the soft palate. The anterior labia are from 12 - 14 mm. in length.

The posterior labia are more prominent, the superior part being known as the torus tubarius. The inferior part of the labia becomes the salpingopharyngeal fold which encloses a muscle of the same name. This fold runs inferiorly, disappearing from view, joining the soft palate. The lenth of the posterior labia are from 14 - 16 mm.

From the lower end of the anterior labium (in a horizontal direction posteriorly) to the torus tubarius is approximately 13 mm.

The anterior and posterior labia gradually converge as they extend superiorly and finally join at their upper extremities.

A calibrated probe can enter the tubes for 10-20 mm. without difficulty and can be visualized clearly by use of a mirror.

The anterior labium of the eustachian tube orifice is 6.5-7.5 mm. posterior to the posterior end of the inferior turbinate.

It is 12-15 mm. from the posterior ends of the inferior turbinates to the posterior labia.

The lower borders of the inferior turbinates, if extended posteriorly, would cross the superior third of the eustachian orifices.

Both eustachian tubes of the first three and the fifth patients discussed are patent. The fourth patient has a patent tube on the left and a scarred, hypertrophic, occluded tube on the side of the lesion. The occluded tube does not change or open with any type of voluntary or involuntary movement and is readily seen to be a pathological eustachian tube.

SUMMARY

Five patients, with large anatomical defects, enabling direct visualization, direct mirror examination and study of the soft palate, nasopharynx and the lumens of the eustachian tubes, are presented and discussed. Special attention was given to the study of the movements of the palate and the patency of the eustachian tube.

CONCLUSIONS

1. The normal eustachian tube is open at all times.

2. No voluntary movement closes or occludes the eustachian tubes.

512 Physicians' and Surgeons' Bldg.

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XL

OTOSCLEROSIS IN IDENTICAL TWINS

FIVE CASE HISTORIES

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NEW YORK, N. Y.

In 1942 I reported my findings in three pairs of identical twins with otosclerosis[1] and reviewed the histories of six pairs reported by others. Since then I have also investigated four other pairs of supposedly otosclerotic twins, but with one exception could not obtain satisfactory examinations. Since then also I have repeatedly tested many of the 150 twins in the Public Schools of New York City. No progressive deafness or positive family history has been discovered.

In clinical practice and at autopsy otosclerosis is a common finding,[2] but in identical white twins otosclerosis appears to be rarely encountered, and as far as I know no otosclerosis has been reported in identical negro twins. Can its rare occurrence in identical twins be predicated on a halving of the inheritance factor?

Of the more than 200 twins whom I have examined 75 per cent were adolescents (a low incidence epoch for otosclerosis), so that no reliable inference can be obtained from the failure to diagnose a single case of otosclerosis in this age group. A similar number of non-twins might also reveal no deafness from otosclerosis. However, the fact that in all but one of the five pairs of twins here reported the deafness was not only bilateral in every individual but was shared by both of the twins would seem to imply no halving of the potency of the inheritance factor.

In order to conserve time and space instead of reporting individual histories and examination findings I shall stress certain pertinent agreements and differences in the five pairs of twins here reported. The fourth pair will be discussed more in detail because it presents a unique picture in several aspects.

The history of the twins in infancy, childhood, adolescence and maturity as to health, habits, and the dates and severity of diseases

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suffered, also the diets, likes and dislikes were carefully investigated. With few exceptions, to be noted, there were no impressive differences found.

In every instance the twins were nursed or fed similarly and their mother's health and breast milk were shared by both alike. Each pair of twins lived closely together, usually sleeping in the same bed until married. The fingerprints and the photographs were remarkably similar in each pair.

In addition to the personal and family histories, otological and laboratory examinations and repeated hearing tests were made by air and bone conduction.

In studying the mothers' histories, (Table 1), it was noted that the family history of deafness was negative in Twins 1 and 5. The outstanding observation is that in every instance lactation was probably deficient. In two instances (1st and 4th pairs) there were two or more siblings born before the twins and in only one instance (2nd) was a sibling born after the birth of the twins.

The twins weighed approximately the same at birth in three pairs and differed only by ½ pound in two pairs. In every instance menstruation began earlier in the twin now the deafer of the two (Table 2).

In one pair (2nd) the deafness was first discovered in the now less deafened twin. Tinnitus is more marked in three pairs in the twin with the greater deafness. It was also found in two pairs in the twin with the lesser degree of deafness. Tinnitus was present in at least one ear in every pair of twins. In three pairs there was hypertrophied lymphoid tissue in the nasopharynx in one twin, the one with the greater deafness. In the eight females four of the twins married and three have borne one or two children with no increase in deafness.

The close agreement in most of the data on the blood examinations is striking (Table 3). However, with one exception (1st pair) the white blood count is lower in the deafer twin.

In regard to the blood chemistry (Table 4) the cholesterol estimations were close for both twins except in the 5th pair (the deafer twin having the higher amount). The calcium and inorganic phosphorus were very close, except in the 4th pair in which the deafer twin had less. In three pairs the phosphatase activity was lower in the deafer twin, and with one exception (4th pair) it was lower in

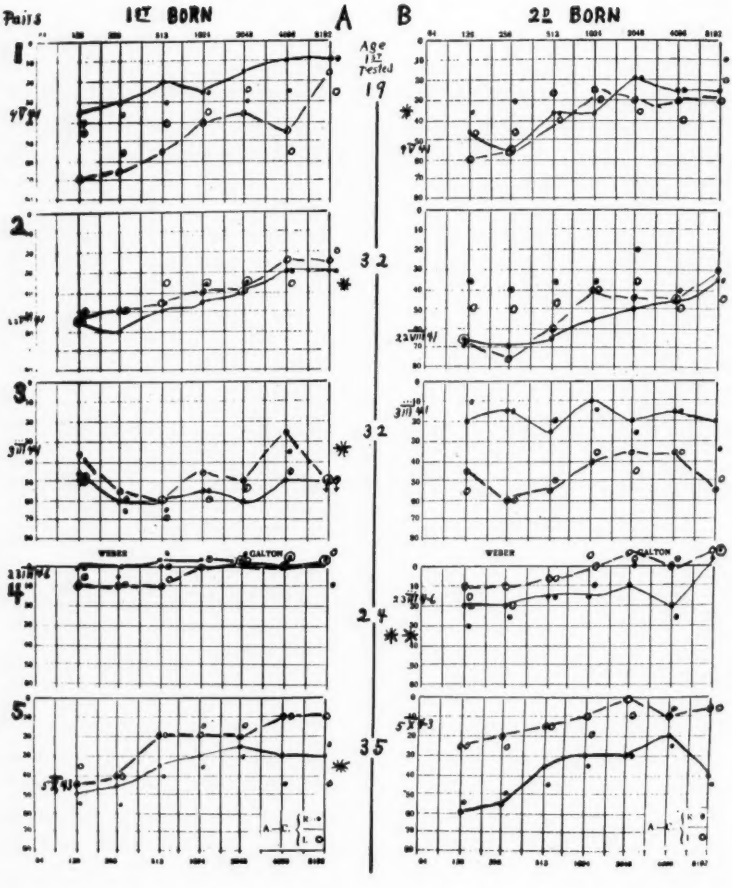

Fig. 1.—Audiograms of ten identical twins, arranged in pairs, Twin A, the first born, being on the left; Twin B, the second born, on the right. The dates of the first tests are given and plotting is directly on the ordinates at each frequency. The dot represents the right ear and the circle the left ear. The last tests, made in 1946 and 1947, are shown by the same symbols plotted just to the right of the ordinates at each frequency. The twin in each pair who has taken sodium fluoride, gr. 3/1000000, is indicated by an asterisk. The twin that is taking a tablet containing fluoride, gr. 1/500, calcium phosphates and vitamin D is indicated by two asterisks.

the twin who first noted any deafness. It is not known what, if any, significance can be attributed to the low phosphatase activity. It is not an infrequent finding in otosclerosis patients. The basal metabolic rate was in close agreement except in the 1st and 5th pairs. The deafer twin in three instances has weighed the more since early childhood. In the 2nd and 4th pairs the twins are now practically identical in weight. With the exception of the 5th pair all of these twins are females.

Figure 1 shows the audiograms of the 10 twins arranged in pairs. In every case, except the right ear of twin B, 5th pair, bone conduction is normal and except for normal hearing in twin A, 4th pair, the deafness is bilateral and shared by both twins. Guild's percentage of bilaterality found at autopsy was approximately 67%. The sampling is too small for deductions as to the meaning of this high ratio of sharing the deafness by the two ears of the individuals, or by the two twins in each pair. In every pair the deafer twin was tense and nervous; the better hearing twin more normal.

It will be noted that the deafer ears in each pair show little difference in hearing levels. Both the better and the poorer ears show little if any loss since the first examination. In twin B of the 1st and 2nd pairs the marked improvement since the first examination was due to the clearing up of upper respiratory infections, In the 4th pair the hearing loss is so small in either twin that obviously there could be little difference between their hearing levels. It is noteworthy that except in twin A of the 5th and oldest pair the high tones have suffered little or no further loss during several years. This is a common finding in uncomplicated otosclerotic deafness.

One twin in each pair has taken sodium fluoride, gr. 3/1,000,000, once to thrice a day. In one pair one twin is taking the fluoride, gr. 3/1,000,000, and the other a tablet containing fluoride, gr. 1/500, with calcium phosphates and vitamin D. Fluorine was administered because of its apparently beneficial effect in the prevention of dental caries, and the other minerals and vitamin D because of their importance in the metabolism of bone. The teeth and the petrous capsule being the two densest and hardest calcium tissues in the body it seemed plausable to believe that to maintain their health, these tissues require a more ample supply and better assimilation of mineral salts than other tissues. It would appear that in all who took flourine the low tone loss in both ears has been stabilized, and therefore we hope also the lesion causing the ankylosis. Too few years have elapsed to determine the efficacy of our treatment.

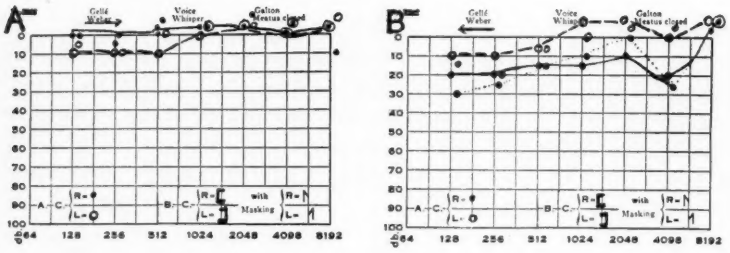

Fig. 2.—Audiograms of the 4th pair of twins, made March 1946 and March 1947.

Slight changes in the audiograms occurred from time to time as would be expected even in people without otosclerosis. Repeated testings are required to insure reliable interpretations. As in all patients showing any tendency to low calcium blood serum content, the calcium intake was increased and the diet supervised. When the acid phosphates were low, phosphorus intake was also increased.

The thing that has surprised me most over the years is the tendency in the great majority of cases for the lesion to stabilize and this in spite of child bearing. In other words, they have suffered little or no further loss in hearing in spite of the otosclerosis.

In the 4th pair of twins I am able to diagnose otosclerosis in twin B but not in twin A. This suggests a greater possibility than usual of finding differences in the environmental factors which might affect the etiology or cause of the otosclerosis. The first tests were made in March 1946 and the last tests were made in March 1947 (Fig. 2). In this pair of twins the following observations are notable:

No deafness was admitted by either twin until after my examination.

There was a history of otosclerosis in the father and of progressive deafness in several members of his family. There was no deafness in the siblings.

In the affected twin (B) the hearing loss was slight by ordinary standards but not when one considers the high normal hearing for all of the octave frequencies over 512 in this individual.

In the apparently as yet unaffected twin (A) the hearing was average or high normal for most of the audiometric scale but a slight elevation of the threshold is evident for the lower frequencies. In view of the positive family history and the deafness in twin B, should we suspect a subclinical lesion in twin A and take steps to stop it? In my opinion the answer is "yes".

The otoscopic and medical examinations showed close agreement. Twin B is tense and nervous. The tonsils and adenoids were removed at 13 years of age.

The laboratory data show little if any significant differences between the twins. There is a microcytosis and hypochromia in twin B.

The mother nursed the twins for only three months because her milk was insufficient. (Deficiency in the mother's milk is common to all of the twins.)

The calcium content of the blood serum is low normal in both (twin A, 9.2 and twin B, 9.6). The difference in favor of twin B may be because she now drinks more milk than twin A.

A high calcium content does not necessarily imply that the calcium is normally assimilated or utilized by the tissues. During gestation, lactation and childhood any deficiencies which may have been present in nutrition, chemical intake and utilization, and any endocrine and emotional factors are later more or less stabilized within normal limits and our tests do not show prior deficiencies.

The sclera of twin A (nonotosclerotic twin) were white. The sclera of twin B (otosclerotic twin) were markedly blue, similar to those found in individuals with brittle bones (osteogenesis imperfecta, fragilitas ossium). The degree of blueness varies from time to time. There was no history of fracture in these twins. Grading the blueness of scleras into five degrees, from 0 for clear white to 5 plus for the most marked, the sclera in twin B is estimated to be 4 plus. The degree of blueness is judged before a diagnosis is made, in fact before testing, to avoid being influenced even subconsciously by the diagnosis.

Twin A has been taking fluoride, gr. 1/1,000,000, twin B fluoride gr. 1/500, with calcium, phosphates and vitamin D. The diet and especially its mineral content have been carefully supervised. The loss of hearing in the right ear of twin B appears to have slightly increased for the low tones (as shown by the dotted lines) since the

first examination. The hearing in twin A shows no change. Twin A has recently married. The deafness, the blueness of the sclera, the thyroid nodule and the nervous disposition found in twin B were the outstanding differences in this pair of twins.

SUMMARY

The pertinent agreements and differences in five pairs of identical twins with otosclerosis are reported. The following observations were discussed:

1. The mothers' milk was deficient in every instance.

2. In the eight females menstruation began earlier in the twin who is now the deafer in each pair.

3. Medication containing fluorine, and the intake of calcium and phosphorus in the food was prescribed for at least one twin in each pair.

4. During several years little if any increase in deafness has been noted in these twins in spite of several childbirths and upper respiratory infections in several instances.

5. With the exception of one individual (twin A, 4th pair) the deafness was shared by both individuals in each pair and by both ears of both individuals in each pair. This is interpreted as indicating that in these twins either the hereditary or environmental factors, or both, must have had similar etiological effects in every pair but one. In this pair only one twin had deafness, blue scleras and a noticeably nervous disposition. The other twin had normal hearing, white scleras and no noticeable nervous symptoms. This exceptional pair of twins will be followed with interest in the years to come.

If we are able to obtain even a few pairs of twins like this with only one of the twins affected by the deafness, it is felt that something of value may be discovered in the etiology, prevention and arrest of otosclerosis.

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TABLE 1.—MOTHER'S HISTORY.

Twin		AGE OF TWINS AT 1ST EXAM.	FAMILY HISTORY OF DEAFNESS	HEALTH DURING PREGNANCY	LACTATION	DIET IN PREGNANCY	DRUGS	NO. OF SIBLINGS BORN BEFORE	NO. OF SIBLINGS BORN AFTER
1 F	A	19	Neg.	Very ill	3 mos.	Mostly milk and ice	None	9	0
	B								
2 F	A	32	Pos.	Normal	Scanty	Normal	None	0	1
	B								
3 F	*A*	32	Pos.	Nausea	Nil	Normal	None	0	0
	B								
4 F	A	24	Pos.	Good	3 mos.	Normal	None	2	0
	B								
5 M	A	35	Neg.	Good	?	Normal	?	0	0
	B								

A is first born twin; B second born.
Letter italicized indicates the deafer twin.

TABLE 2.—SIMILARITIES AND DIFFERENCES IN TWINS.

		AGE	F.H. OF DEAFNESS	WEIGHT AT BIRTH	MENSES AT	AGE DEAFNESS FIRST NOTED	EATS MORE OF	TINNITUS	EXANTHEMATA; MUMPS	COLDS	EARACHES OR DISCHARGE	LYMPHOID TISSUE IN NASOPHARYNX	ALLERGIES	LIVING PROXIMITY	MARRIED	CHILDREN
Twins																
1 F	A	19	Neg.	4½	15	18 L	Fish spnch	Now	Same	Few	No	Little		Same bed B near window	+	1
	B			4	14	12 L	Salt, milk	Slight				More			+	1
2 F	A	32	Pos.	2½	16	18 R	Milk	No	Same	More	No	More	AM clog-ing	Same bed until B marr. 23	0	0
	B			2½	14½	21 L	Gr. vegs.	Yes		Few		Little			+	2
3 F	*A*	32	Pos.	4	13	20 L		Yes	Same	Many at 26	1	Yes		Same bed A near window	0	0
	B			3½	14	20 L	Milk	No		Few	No	Neg.			0	0
4 F	A	24	Pos.	7½	14.1	Never	Fruits	Neg.	Same	Same	No	Neg.		Always to-gether	+	0
	B			7¾	14	23		Slight							0	0
5 M	A	35	Neg.	8		34	Same	2 plus	Same	More	No	Neg.		Same until 27 yrs.		
	B			8		19										

Letter italicized indicates deafer twin.
A is first born twin; B second born.

TABLE 3.—BLOOD EXAMINATIONS

	Pair 1		Pair 2		Pair 3		Pair 4		Pair 5	
Blood Count:	A	*B*	A	*B*	*A*	B	A	*B*	A	*B*
Hgb.	82%	83%	74%	85%	82%	80%	88%	78%	103%	100%
R.B.C.	4350M	4600M	4420M	4450M	4480M	4700M	4540M	4280M	5600M	5000M
W.B.C.	6,100	7,700	6,200	6,000	6,400	8,100	10000	6,800	5,750	5,400
Col. Index	.95	.90	.84	.96	.93	.85	.97	.92	.91	1.0
Corpuscles	Norm	Norm	*	Norm	Norm	Norm	Norm	**	Norm	Norm
Polys, seg.	47%	58%	40%	50%	58%	64%	49%	55%	47%	43%
Polys, non-seg.	2%	3%	0%	0%	2%	3%	12%	7.5%	2.5%	4.5%
Lymphs.	46%	34%	54%	44%	34%	30%	30%	28.5%	40.5%	48.5%
Monos.	1%	2%	5%	4%	5%	3%	8%	8%	6.5%	8. %
Eosins.	4%	3%	1%	2%	1%	0%	1%	1%	1.5%	0. %
Basos.	0%	0%	0%	0%	0%	0%	0%	0%	2. %	0 %
Other cells	0%	0%	0%	0%	0%	0%	0%	0%	0 %	0 %

*Some anisocytosis and microcytosis.

**Microcytosis, hypochromia.

A is first born twin; B second born.

Letter italicized indicates deafer twin.

All twins are females except Pair 5.

TABLE 4.—BLOOD CHEMISTRY AND BASAL METABOLIC RATE.

Blood Chemistry:	Pair 1		Pair 2		Pair 3		Pair 4		Pair 5	
	A	*B**	A*	*B*	*A**	B	A	*B**	A	*B**
Chol.	210.0	200.0	215.0	200.00	280.0	300.0	151.0	166.0	134.0	201.0
Calcium	10.1	9.8	9.2	9.3	9.7	9.8	9.2	9.6	9.9	9.5
Inorg. phos.	4.4	4.3	4.0	4.4	3.21	3.41	5.6	4.1	4.1	5.
Phtse. Act.	4.9	3.7	1.6	2.3	2.8	3.2	1.5	3.0	3.9	2.7
Wassermann	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.
B.M.R.	—7	+7	—6	—1	+2	+7	—5	—9	—7	—17
Height	63¾	63½	62½	61¼	64½	64¼	62¼	63¼	68¼	68
Weight	103½	113	108	108	127½	117	115	114½	143¼	171
Pulse rate	80	70	76	76	80	88	68-62	58	64	62

*Indicates twin who first noticed deafness.

A is first born twin; B second born.

Letter italicized indicates deafer twin.

All twins are females except Pair 5.

XLI

TESTS FOR LABYRINTH FUNCTION FOLLOWING STREPTOMYCIN THERAPY

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The selective effects of streptomycin on the labyrinth and especially its predilection for the vestibular system are for the otologist particularly interesting phenomena.[1, 2] Of the many drugs said to have an effect on the ear, quinine is perhaps the best known. There are other poisons which affect either auditory or vestibular function or both, but salicylates, nicotine and perhaps barbiturates may be considered as next in importance to quinine and streptomycin. Dosage and idiosyncrasy are the dominant factors in the occurrence of toxic reactions from all of these drugs.[3, 4]

The opportunity to study thoroughly the auditory and vestibular systems of 32 patients at various periods of streptomycin therapy presented itself to the authors when a program sponsored by the American Trudeau Society was started at Laurel Heights Sanitarium, Shelton, Conn., to determine the efficacy of this drug in the treatment of pulmonary tuberculosis. In addition the authors have had the good fortune to see more than 90 cases of vestibular dysfunction following streptomycin therapy at Columbia-Presbyterian Medical Center, Halloran General Hospital,[5] New York Hospital, Mayo Clinic, Summit Park, Underhill, Cedarcrest Sanatoria, the New Haven Hospital and the Veterans Hospitals at Sunmount, N. Y., and Newington, Conn. Only 11 cases of deafness due to the drug were seen in these hospitals and only three of these were not associated with either tuberculous or influenzal meningitis. Inci-

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dentally none of these three patients had excessive dosage or renal dysfunction. Compare McDermott.[6]

The report which follows is concerned largely with the otological aspects of streptomycin therapy as observed at Laurel Heights. Here each patient received a careful otic examination, including neurological, audiometer and caloric tests, before therapy was begun. Fifteen patients were given 1.8 gm. per day in divided doses every three hours subcutaneously. Thirteen received 2 gm. for one to four months. The remaining patients had received the drug for less than one month at the time of this report and are therefore eliminated except when discussing normal reactions.

Audiometer tests were done every two weeks during the period of treatment. These tests were made with an Audio Development Company discrete frequency audiometer, standardized by the U. S. Bureau of Standards. Both air and bone conduction readings were made in all preliminary tests. Subsequent readings were for air conduction only unless a hearing loss was noted, in which case bone conduction readings also were made.

Since it was not practicable to have a room completely insulated from sound, an attempt was made to approximate one. This was accomplished by choosing a room high up in the building as far away from noisy spots as possible and yet readily accessible. Inside the room, a framework approximately 7x9x7 feet was constructed and covered with a double layer of blankets inside and outside; carpets covered the floor. A General Radio Sound Level meter registered a noise level of 24-28 db. inside the room. Measurement of ambient noise outside the framework and with the doors open indicated an attenuation of 10-15 db. for such noises as a nearby elevator. This is an easily constructed, inexpensive audiometer room and is satisfactory for the type of clinical observation needed for this work. Of course, outside noise was at a minimum so the noise inside the room was also low.

All but one of the 23 patients who had had more than two months of streptomycin therapy maintained a normal audiogram. This one patient who did not showed a 65 db. loss for the tones 2048, 4096 and 8192 (Fig. 1). She had received 1.8 gm. of streptomycin per day for 121 days. Incidentally, this patient had no caloric responses before, during, or after her streptomycin treatment. She was a demerol addict but the audiograms were taken after the patient was off demerol. She did not have syphilis or other disease except severe pulmonary tuberculosis.

AIR CONDUCTION

DATE	RIGHT							LEFT						
	128	256	512	1024	2048	4096	8192	128	256	512	1024	2048	4096	8192
Nov. 23	10	10	20	5	0	0	—10	10	0	10	5	0	—5	—5
Dec. 4	10	10	10	5	0	0	—10	10	5	10	0	—5	—5	—5
Dec. 20	15	5	10	5	0	—5	—10	10	0	0	5	—5	10	25
Jan. 3	15	10	10	5	—5	0	5	5	0	0	5	0	0	20
Jan. 30	15	10	5	5	0	25	50	15	10	5	5	—5	25	60
Feb. 21	30	20	15	5	5	50	50	15	5	10	5	10	45	50
Mar. 7	15	15	10	5	40	40	40	15	20	5	15	40	55	55
Mar. 31	20	20	15	20	60	50	40	25	25	15	25	55	50	55

BONE CONDUCTION

Mar. 31	30	30	30	45	60	....	....	20	15	40	45	60	....	....

Fig. 1.—Audiograms of patient who lost hearing acuity following streptomycin therapy—1.8 gm. per day for 121 days beginning November 24, 1947.

In contrast to the lack of auditory symptoms almost all of the patients had vestibular symptoms and signs. Daily checks were made for spontaneous and positional nystagmus and a few days before vertigo was noticed by the patient a fine nystagmus could be made out with lateral gaze in either direction. It is presumed that this is due to lack of tone in the eye muscles from poisoning of the vestibular apparatus or its connections. One of the first symptoms noticed by the patients is blurred vision while reading.

Spontaneous nystagmus of second and third degree did not occur in this group of patients, although it has been occasionally observed in the larger group mentioned above, especially in persons with tuberculous meningitis. As a matter of fact the vertigo following streptomycin therapy is not an ordinary vertigo. The patients complain that when they turn over in bed they feel as though they were continuing to turn. Even on turning the head alone they feel as though it were continuing to turn. Often they actually do turn their heads further than they intended. The vertigo may be so severe as to induce nausea and vomiting but on the whole if the patients do not turn their heads there is no vertigo. Later on there is vertigo only on sudden turning. At first there is often overreaching when trying to pick up an object such as a glass of water.

The time of onset of vertigo is interesting. Vertigo began in 2 patients in from 11 to 20 days; in 11 patients in from 20 to 25 days; in 3 patients in from 30 to 40 days and in 1 in from 50 to 60

days; 6 did not complain of vertigo at any time. The subjective vertigo lasted about 5 days in 4 patients; about 10 days in 5; about 15 days in 3; about 25 days in 2; about 30 days in 1, and about 50 days in another. It was accompanied by nausea in 2, nausea and vomiting in 5. All but six were bed patients making it impossible to perform Romberg's test. These six showed marked positive Romberg reactions after 25 to 30 days.

The vestibular dysfunction could best be brought out with vestibular tests. All but three of the 23 patients who had received enough streptomycin at the time of this report to be adequately studied developed loss of reaction to rotation and caloric stimulation when the treatment was continued more than 25 days. It was deemed advisable to study this loss of vestibular effect with some care in an effort to find out if it was peripheral or central in origin and also to see if streptomycin-treated patients might throw more light on the problems of the vestibular mechanisms in general.

The standard rotation tests with hand control of the Bárány chair (10x20 seconds and 10x10 seconds) are too gross to be of much help and are contraindicated in most patients sick enough to need streptomycin.

Caloric tests can be done in bed or on a stretcher and are therefore more suitable. There are two types: the mass or volume methods, after Bárány, and the minimal methods, after Kobrak. Bárány used 100-200 cc. of water at 68° F. with the head in the upright position. Kobrak used 5-10 cc. of water varying between 95° and 85°, the amount being increased from 5 to 10 cc. and the temperature being decreased as needed to elicit the labyrinthine threshold.

The Kobrak minimal method was modified by Demetriades and Mayer as follows: head upright, 5 cc. of water at 55° F.; after three to four minutes if no reaction occurs the head is bent 60° backwards, "the optimal position," and an additional 10 cc. of water is injected into the external auditory canal.

The Atkinson modification is: 1 cc. of ice water injected into the external auditory canal with the head tipped to one side and the canal being emptied at the first sign of nystagmus. The latent period and the duration of nystagmus are noted. The average latent time is 15-60 seconds while the duration of the nystagmus is normally four times the latent time.

Fleischman made large scale examinations by the minimal method varying the temperature, volume and duration of irrigation. He found no significant difference no matter how he did the test. There was still 15-30 seconds' latent time and 60-120 seconds' duration time for the nystagmus.

Ruttin used 150-200 cc. of cold water, obtaining an average of 15-30 seconds' latent time and 90 seconds' duration time.[7, 19]

Any one of these methods will produce fairly satisfactory qualitative results if one carefully follows the same routine for each test and learns to interpret the results; however, none proved accurate or feasible enough to study the finer aspects of streptomycin effect.

In seeking a caloric test which embodies the advantage of minimal stimulation yet lends itself to ease of control because of sufficient volume Fitzgerald, Cawthorne and Hallpike[8] developed the following method: A two-pint metal douche can was filled from hot and cold taps. The temperature was adjusted first to 30° C. and then to 44° C. This will produce a minimal stimulus in opposite directions, for when the water reaches the ear it is presumably 7° C. above body temperature for the hot and 7° C. below normal body temperature for the cold. Each ear is tested with water at both temperatures. The tube used in the original experiments was more than five feet long, the nozzle tip was said to be 4 mm. in diameter, delivering about 240 cc. in the 40 seconds used as the stimulation time. The water level was approximately two feet above the ear being tested. The nystagmus was timed with a stopwatch from the beginning of the irrigation. Timing was begun at this point to eliminate the error which occurs when one tries to decide when the nystagmus has really commenced. Timing from the beginning or end of stimulation means timing for only one variable, the end point of the reaction. Observation of the nystagmus is made without accessories, such as Bartel's glasses or the Frenzel Leuchtbrille in a dark room. We agree with Hallpike et al. that these seem to make the end point even harder to evaluate. When the nystagmus is checked by two observers simultaneously who are watching the conjunctival vessels for movement and taking care to do the test within 15 seconds of testing the temperatures, a fairly consistent end point is said to be obtained with practice.

The subject is asked to fix his vision on a suitable mark on the ceiling or wall while lying down with the head raised 30°. This is maintained with a backrest allowing an angle of 30° beginning

Fig. 2.—Set-up for doing caloric tests according to modified Fitzgerald-Cawthorne-Hallpike technique.

at the waist; thus the horizontal canal is placed in the vertical or optimum position. Hallpike et al report that normal readings with the cold water ranged between 90 seconds and 130 seconds in 80 per cent of the cases, were 80 seconds or 140 to 160 seconds in the remaining 20 per cent.

The authors found that there was considerable variation in temperatures using a two-pint douche can and long tube. Consequently the following minor modifications were made: the two-pint metal douche can was replaced by a gallon thermos jug of the common picnic variety making it possible to maintain a constant temperature for about 5 minutes with the hot water and 30 minutes with the cold. Only 30 inches of rubber tubing was used. To be sure of the temperature of the water which reached the ear, a thermometer was placed in the tubing within six inches of the nozzle. The author's nozzle measured 2.5 mm. inside diameter. With an ordinary two-pint douche can and six feet of tubing the temperature loss at the ear in a room temperature of 24° C. was 1.5° to 2° in a period of two minutes. The loss with the vacuum cans and the 30-inch tube was 0.1° to 0.2° for water at 44° C, but even this was eliminated by taking the temperature next to the ear. The temperature loss for water at 30° C. was 0.2 to 0.5° with the uninsulated

pint can but was constant for one-half hour with the vacuum bottles. Insulation of the tubing was found not to be essential.

To facilitate doing repeated examinations on numerous subjects, two jugs, one with water at 30.1° C. and one with water at 44.1° C., were suspended on a stand fastened to rollers. Two patients on movable stretchers were placed head to head with the stand between them (Fig. 2). With this set-up the patients could be alternated, thus allowing one labyrinth to recover before the other on the same patient was tested. Alternating patients makes it possible to do caloric tests on about four patients per hour.

The stimulus time and the total time are kept respectively by the observer and an assistant independently, the assistant receiving start and stop signals from the observer. The time is recorded graphically, after the method of Hallpike (Figs. 3 and 4).

Ten patients showed a variation of less than 10 seconds for both hot and cold in either ear. In 10 the cold reaction was longer by 10-20 seconds. In one case the reaction to cold was 40 seconds longer than the reaction to hot in both ears. In 8 the hot was longer by 10-20 seconds. In 1 the hot was 30 seconds longer and in 1 the hot was 40 seconds longer. In one case there was no reaction to hot or cold. This was in the demerol addict.

The finding of similar reactions to both hot and cold is somewhat different from the observations of Fitzgerald, Cawthorne and Hallpike.[8] These authors, in using both hot and cold, found the cold reaction to exceed the hot by about 10-15 seconds in 80 per cent of the examinations. The authors believe that their own finding of nearly similar reactions to both hot and cold water is due to using the thermometer close to the ear, thus making certain the hot stimulus is not reduced because of decrease in temperature.

One other point of this study was to establish the time after beginning streptomycin therapy that a significant change took place in the vestibular apparatus. This would have clinical application in deciding how long the drug can be given before a change will occur. The problem of dosage level did not occur in this series because the dosage level was essentially 2 gm. per day for all patients. Analyzing the results of the caloric tests revealed the following: In 25 patients whose labyrinths were tested after streptomycin was begun a shortening in the nystagmus time of at least 30 seconds was found on the 20th to the 25th day. It is possible that a few patients had a shortened time before the 20th day, for many

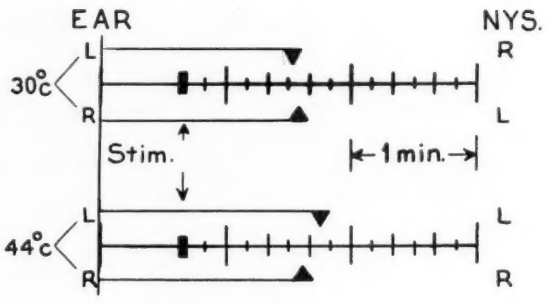

Fig. 3.—Normal Calorigram before treatment.

were not tested between the 14th and the 20th day and some of them had subjective vertigo before a shortening or absence of nystagmus time had been established.

Considerable change occurred also in the nystagmus itself. There was decreased activity, with intermittent beats which were jerky and of inconstant amplitude as compared to the normal in the same patient. It is here that a mechanical or electrical nystagmogram would be valuable for comparison as to amplitude, regularity and number of beats per second. It is nearly impossible to compare the finer changes by visual observation. An attempt was made to qualify the nystagmus by indicating the normal activity as 4 plus and decreasing to one plus for a barely perceptible movement. Over 200 such examinations were done on 30 subjects. One patient had 29 caloric tests done on each ear, 6 with water at both 30° and 44° C., the remainder with 30° C. water only, in addition to several with ice water and water at 20° C. Four were tested every two days, 4 every seven days and 14 at two-week intervals. Further analysis reveals that 20 subjects failed to respond to any labyrinthine stimulation following streptomycin therapy at the following times: 5 between the 20th and 30th day; 3 between the 30th and 40th day; 2 between the 40th and 50th day; 8 between the 50th and 60th day; 1 on the 67th day. Three still gave responses after 121 days, although they showed irregular nystagmus time for both hot and cold stimulation and a definite decrease in activity.

The question comes up as to whether there was at any time a hyperactivity. The caloric time at 10 days appeared slightly longer

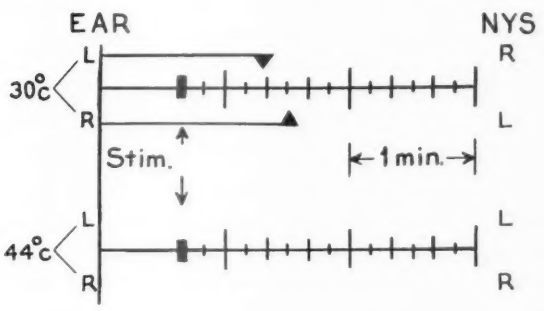

Fig. 4.—Calorigram showing deficient reaction to cold and absence of reaction to hot after two weeks of streptomycin therapy.

than the pretreatment time in a number of cases but it is questionable without more data whether this is significant.

It will no doubt be remembered that Fitzgerald and Hallpike showed a range between 90 and 130 seconds in 80 per cent of normal subjects, while in 83 per cent of the authors' series there is a range of 110 to 140 seconds, a difference of 40 seconds in Hallpike and Fitzgerald's series and 30 seconds in ours. It is possible that the narrower margin in our series is due to more exact temperature control.

It would be a tremendous help to make electrical recordings of the nystagmus in a group of normal subjects as well as streptomycin-treated patients to compare the time and the amplitude against that obtained by visual methods. Anyone who has done many vestibular tests will testify that reading the end point is a most difficult task. The authors believe this difficulty is the cause of the discrepancies shown above. With the elimination of guesswork in regard to the temperature delivered to the ear by the use of a stimulus that is timed precisely and of sufficient quantity to give a definite stimulation, it should be readily seen that the one great variable in the technique is reading the end point. We hope to overcome this by the use of an adaptation of such standard apparatus as an electro-encephalograph or an electrocardiograph machine. This has been done experimentally[9] and there is no reason why it can not be done clinically. Rotation with measured acceleration would be even more accurate, hence should be done in experimental studies. Nothing is said about variables such as fixation, voluntary eye and

lid movements, lack of interest, instability, or distraction. Any one of these will change the nystagmus time, so that with all the care being taken to procure an exact technique a variation of 30 seconds is obtained between different tests. These variables should all be materially reduced by blindfolding the patient and taking electrical recordings.

The gait of all patients who were able to walk was studied at various times during and after treatment. All who lost caloric response walked with more or less broad bases and some ataxia. This was very apparent after three or four weeks of treatment, but gradually became compensated so that after four months incapacity was not perceptible if vision was unobstructed and the walking surface smooth. If the patients walked on a graded lawn, whether on the flat or a slight slope, the gait became unsteady and the base broadened perceptibly. During some tests mattresses were placed on the floor to protect against injury if anyone fell. It was noticed that walking on these mattresses was very difficult even when vision was used. It seemed as though the stimulus was delayed. Imbalance would occur but as a rule recovery would intercept an actual fall; when blindfolded the patients were unable to walk more than a few steps on the lawn or the mattress without falling. This was true for all patients, even those who had compensated the most.

When the blindfolded patients walked on a smooth hard surface, the gait they assumed depended to a large extent on the length of time since the loss of vestibular response. Walking, even without a blindfold, was next to impossible in the interval between the beginning of a changed response to caloric stimulation and final occurrence of a negative response. As time passed, the patient became, as a rule, quite steady with only a slight stagger and a broad base until three months after a negative response to caloric stimulation first occurred when they did well under ordinary conditions.

All streptomycin-tested patients who had tried walking in the dark in unfamiliar surroundings complained of severe difficulty in maintaining balance. A good example is trying to change seats in a dark theatre.

It will be extremely interesting to follow these patients and note if they continue to compensate for the loss of vestibular function. There is a question, of course, whether the loss is permanent, but so far there has been no recovery in our cases up to four months subsequent to the first negative caloric response. We have seen 8 patients elsewhere who have not compensated one year after therapy.

The younger patients seem to compensate more easily than the older patients. Some patients over 60 years of age did not develop adequate compensation in the four-month period of observation. This is a fact worth considering when streptomycin is projected for patients in the older age group.

Rotation tests were carried out in 7 subjects; all were rotated 15 times in 15 seconds with eyes open and closed. It made little difference how fast, how many times, whether or not a blindfold was used; when stopped suddenly they stood up and walked a straight line with no effort whatever. Four normal subjects were tried at the same speed and number of turns, with the usual results of marked dizziness and inability to walk straight immediately after the stimulation.

Rotation had not the slightest effect on the patients who had recurrent heavy doses of streptomycin. They showed normal Romberg tests, no past pointing or ataxia of any description after the stimulation, and no nystagmus.

It has been stated already that Romberg and past-pointing tests were done at various times during treatment and that compensation had taken place, so that in four months the Romberg and past-pointing tests were essentially normal.

Another apparatus for studying the labyrinthine function of these patients is the tilt-table. Tilting out of the horizontal plane about a horizontal axis has been used on animals for some time. The slow tilt is said to test otolithic function[10] and the quick tilt to test the semicircular canals.

Ewald and Thomas found that after removal of one labyrinth if the animals were quickly tilted to the operated side they were easily upset; Tait and McNally[12] first used this test on patients in 1926. They showed that in the absence of both labyrinths a quick tilt in any direction would easily upset the patient and concluded that this was due to an ablation of the semicircular canals.

Rademaker and Garcin[14] adopted the quick tilt test in the clinic as an aid in diagnosing loss of one or both labyrinths. In the main they confirm the above conclusions of McNally. Radmark[15] found that placing the patient on hands and knees and tipping through the patient's longitudinal axis gave the most information.

It was found to be a fairly simple matter to construct a satisfactory tilt-table for about thirty dollars. Even this was more than

necessary because the authors' design included extras which made it collapsible and therefore easily portable. The Glorig table is about 18 or 20 inches high and approximately four feet square. Large mats of sponge rubber are used as pads under the subjects' hands and knees to make it more comfortable.

Unfortunately only eight of our group were physically fit enough to undergo tilt-table tests. In all but one of these there was a bilateral lack of response to caloric stimulation, even to 100 cc. of ice water. They had all received 90 days or more of therapy. There was no hearing loss. All had compensated fairly well and showed at the time of the tilt-table tests essentially normal Romberg and past-pointing tests. Positional nystagmus had disappeared. Walking was excellent unless blindfolded, when all staggered considerably on an even floor and fell when on uneven ground. Rotation (15 times in 15 seconds) produced no nystagmus and no dizziness. The patients were placed on hands and knees first parallel to the longitudinal axis of the tilt-table and then at right angles to this axis. Each was tilted with eyes open and blindfolded. When tilted slowly with eyes open, recovery of a level position resulted, but in place of a smooth coordinated effort as shown by a normal subject, each movement during the adjustment was tardy and when effected was jerky; sometimes the adjustment was insufficient and other times it was excessive. When the tilt was rapid, recovery was accomplished, but as though the message was delayed and when the realization that the table was not level finally occurred, there was considerable scrambling to adjust to the new position. It would seem that reactions to visual stimuli are slower and less exact than to vestibular stimuli. This assumption is upheld by the gait which is discussed above.

When the patients were blindfolded, the following reactions occurred: with the slow tilt there was absolutely no effort to gain the normal position until gravity caused a shifting of the weight. If the angle was held just short of the actual weight shift there was a delayed groping attempt to adjust to the new position. If after a slow tilt to approximately 20° the patient was returned to level very slowly the compensation which had resulted from the tilt remained and the subject held to the attained position when level, as if he thought he was still tilted. If, however, the table was held at the level position readjustment occurred in a second or two, so that if tilting to the opposite side occurred the patient fell to that side when there was only a few degrees of tilt.

Fig. 5.—Quick tilt showing abnormal shifting of the weight after streptomycin therapy. This patient had nearly normal reactions to slow tilt one month after cessation of therapy and three months after loss of caloric response.

If the tilt was rapid, attaining the maximum in about one-third to one-half second, the patient was completely without defense and fell to the lowered side. Seven patients reacted in the same way to both right and left in all tilt-table tests (Fig. 5).

One patient whose labyrinth never showed more than a slightly shortened caloric response, even after 121 days of therapy, was tested with the tilt-table. His reactions seemed perfectly normal.

Radmark[15] has written, "Bilateral labyrinthine destruction causes a total and permanent inability to preserve balance during rapid tipping movements. In cases of pure peripheral vestibular disorders the first phase of the reaction is defective while the final phase displays no pronounced ataxia. If the third phase is pathological, then there is also a central vestibular injury . . . "

If Radmark's views are correct, we could predicate that the streptomycin lesion is probably peripheral, for he feels that if the first phase of the tilt is defective and there is no defect in the third phase the defect is labyrinthine in origin.

On the other hand, one cannot assume from the results of the tilt-table tests that the lesion from streptomycin is localized either

in the semicircular canals or in the utricle alone if we are to accept as correct the statement of McNally,[11] who said, "A slow tilt stimulates the utricles but does not stimulate the semicircular canals, and in response to a slow tilt the utricles elicit a compensatory reaction. A quick tilt stimulates the vertical semi-circular canals to bring about a prompt compensatory reaction of the body musculature."

Also suggestive of peripheral location of the streptomycin lesion is the galvanic test. Galvanic stimulation was performed with electrodes placed over each mastoid. Four normal subjects showed definite subjective vertigo with jerky rotatory and horizontal nystagmus with a current of 3-4 M.A. The nystagmus was toward the cathode in each case.

Four streptomycin-treated patients were tried, all after 121 days of therapy and 24 days after the drug had been stopped. Three showed no labyrinthine response to as much as 80 cc. of ice water. One had a definite although reduced bilateral caloric response in spite of 121 days of treatment. The galvanic stimulation showed the following: the first patient had no nystagmus but complained of considerable vertigo; there was no nausea or vomiting. When standing he complained of inability to keep his balance, that he felt as though his forehead was moist and clammy. This feeling disappeared in a few seconds. The second patient experienced definite vertigo with stimulation and showed slight jerky rotatory and horizontal nystagmus at 4 M.A. The third patient had only slight vertigo at 5 M.A. The fourth whose labyrinth was still active, had a moderate vertigo with slight but definite nystagmus at 4 M.A.

To attempt to locate the lesion and specify its nature up to now is admittedly mere conjecture. Loss of rotation and caloric response with galvanic and tilt-table responses being of the peripheral type leads us to believe the lesion is in the labyrinth. So far we have been unable to find any convincing evidence of pathologic changes in the sections of the labyrinths studied. There seems to be considerable pigment about the neural crests of the semicircular canals, in the macule of the utricle and the scala media; yet we find this also in subjects not treated with streptomycin and are not convinced it is pathological.

Stevenson et al.[16] have found changes in the Nissl body staining of certain cells in the ventral auditory nuclei. We have not yet found them in animal or human material and it is amazing that the changes were found primarily in the auditory rather than the vestibular nuclei. It is hoped that with special stains or with more ma-

terial the answer to this fascinating problem will be found. Meanwhile, it behooves us all to do careful before-and-after-streptomycin hearing and vestibular tests on all patients receiving the drug. We believe the methods presented in this paper are more accurate than those usually employed.

Sound location has been said by Guttrich to reside in the vestibular apparatus.[17] Although we were not primarily concerned with this problem, it would have been a shame to pass up such an opportunity to test this theory. Here were several persons with entirely normal hearing but completely inactive labyrinths. In all of Guttrich's cases there was some loss of hearing and in some it was unequal in the two ears.

Four subjects were chosen whose primary disease allowed outdoor exercise. An open field away from interfering buildings and other objects served as a proving ground. The environment was quiet and there was very little wind. An alarm-clock bell was chosen for sound production. The subjects were blindfolded and asked to locate the sound from a distance of about 50 yards. One of the authors (hearing normal) stood behind the subject not blindfolded, and as the bell rang pointed simultaneosuly with the subject. The arms served as two sides of a triangle with the apex at the bodies. The angle made by both arms was measured and served to calculate the error, if any were present.

The assistant moved from place to place within the 50 yard radius.

This method was not the one used by Guttrich but seemed to serve the same purpose. In all four subjects the angle of error between the subject and the experimenter was less than 5°. During the experiment an airplane flew over. The localization of the ship by the blindfolded subjects was as accurate as by the experimenter. It then seems safe to assume that the vestibular apparatus has very little, if anything, to do with sound location. In this we agree with Diamant[18] who has recently reported some localization of sound in patients with vestibular loss and normal hearing.

YALE MEDICAL SCHOOL

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XLII

CONGENITAL CYSTS AND FISTULAE ABOUT THE HEAD AND NECK

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Because of the unorthodox origin of congenital cysts and fistulae of the head and neck, the information regarding their clinical aspects has not been thoroughly standardized. I feel, therefore, that the report of a case which presents a slight variation from those previously recorded may be of interest. It is universally conceded that all cysts and fistulae of the head and neck, having an epithelial lining, should be treated surgically by extirpation of the entire lining and allowing the cavity to close by first intention. Without a knowledge of their embryologic background, however, one cannot be prepared beforehand for many of the anatomic pitfalls encountered in their management.

Broadly speaking, these anomalies are classified into two main groups: (1) the midline group, or thyroglossal cysts and fistulae, and (2) lateral cysts and fistulae, or those arising on the side of the neck.

There is practically no disagreement regarding the view that thyroglossal cysts and fistulae are derived from the thyroglossal duct, and that this duct is formed by a bud of hypoblast in the midventral floor of the pharynx, growing downward between the mandibular and pharyngeal parts of the tongue, eventually forming the isthmus of the thyroid body and parts of the lateral lobes of the thyroid gland. It has been observed that cysts may form anywhere along the path of the thyroglossal duct from which they are derived, the most common location being immediately below the hyoid bone. Not infrequently, these cysts form in the body of the tongue in front of the hyoid bone. In a smaller number of cases they form between the foramen caecum and the hyoid. In some, they involve the hyoid bone itself. In any location, these cysts present peculiar anatomic and technical problems.

Read before the Sixty-Seventh Annual Meeting of the American Laryngological Association, Chicago, Ill., May 30, 1946.

Lateral cysts and fistulae are even less understood than those of the first group. This is due to a wide divergence of opinion regarding the derivation of lateral cysts and fistulae of the neck. Two distinct and contradictory concepts prevail. One is that these anomalies develop primarily from the branchial apparatus.[1-5] Adherents of this theory are further divided into two groups. One group believes the branchial apparatus in the human embryo is analagous to the gills in adult fish, except that in fish the arches are separated by clefts, whereas in human beings the depressions between the arches represent thin membranes. In rare instances, these membranes are perforated, leaving clefts which persist into adult life as branchial cysts or fistulae. This theory was held by the early investigators, being predicated upon the assumption that there was a fusion of the epipericardial ridges with the lower border of the second and midventral ends of the third and fourth branchial arches, thereby closing the precervical sinus. In refutation of this assumption, it is pointed out that epithelial rests from this source would of necessity be ectodermal in origin, whereas, the histological findings of removed specimens of lateral cervical cysts and fistulae strongly indicate that these structures are of entodermal origin. The significance of this will be brought out later.

For purposes of clarity, I wish to present some material furnished by Frazer, with which he demonstrated that the precervical sinus is formed and disappears without the possibility of its taking any part in the formation of lateral cervical cysts and fistulae.[6] Fig. 2 shows the head of an embryo less than 5 mm. in length. The arches and grooves are shown to be well formed. Their relationship to the position of the heart and their general direction horizontal to the general direction of the body are shown. The otocyst is noted. At this stage, one observes that what is to become the neck of the adult is at a right angle to the main body axis, since the cervical flexure takes place at the second branchial groove. If we now examine the semi-schematic pictures of the lower aspect of the pharyngeal region as given by Frazer, the pericardium having been removed save for its roof, we can understand the relationship of the ventral ends of the branchial arches to each other and to the epipericardial ridge beneath. It is noted that the ventral ends of the first and second arches approach each other and are united, while the ventral ends of the third, fourth and fifth arches recede, leaving a triangular area which encircles the dome of the pericardium. This relationship of the arches to each other and to the pericardium makes it easy to visualize what later becomes the precervical sinus. The pericardium

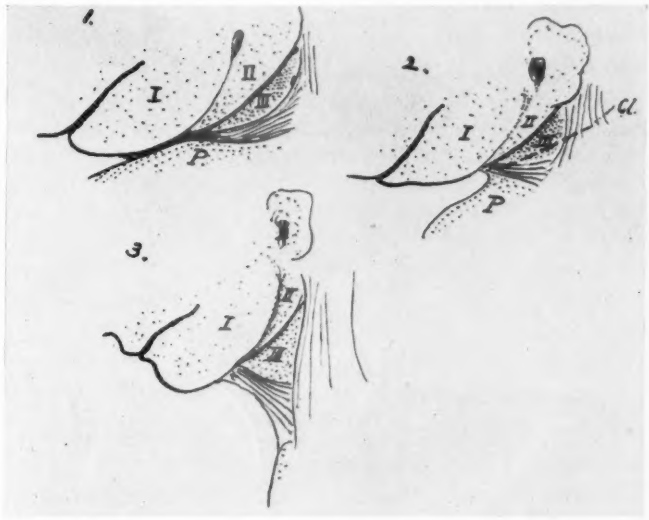

Fig. 1.—Three schemes to show the situation acquired by the third arch as the neck forms. *Cl.* gives the general position and line of the clavicle. (After Frazer).

is below the sinus but does not form its lower boundary directly. There is an epipericardial ridge opposing the outer ends of the arches and forming with those structures the paraxial boundaries of the precervical sinus. A rapid growth of the paraxial boundaries results in a corresponding deepening of the precervical sinus. In the subsequent development, there is fusion of the ventral ends of the first and second arches, the lower border of the second arch thereby becoming the upper border of the paraxial boundary of the precervical sinus. The ventral ends of the third, fourth and fifth arches recede in succession and, as a result, the lateral borders of the paraxial boundaries become more caudal.

From the foregoing facts, the foundation of the neck, with certain fixed points, and its formation in the second and third months can be traced. The first scheme in Fig. 1 shows the general arrangement of the embryonic parts: the ventral ends of the first, second and third arches and the epipericardial ridge. The second scheme shows the same conditions when the clavicular rudiment (Cl) is first present and the sternomastoid is distinct. The third scheme represents the later stage, showing the lengthening of the neck. The infrahyoid

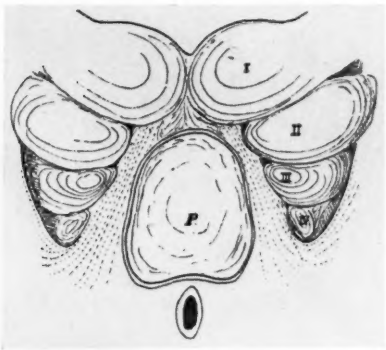

Fig. 2.—Semi-schematic view of the lower aspect of the pharyngeal region, the pericardium having been removed, save for its roof. *I, II, III, IV,* pharyngeal arches, external; *P,* roof of pericardium. The whole region is somewhat flattened out to show the relations. (After Frazer)

muscles are drawn out on the ventral side and fixed to the clavicle and hyoid. From this concept of Frazer's, it can be readily understood that the floor of the precervical sinus ultimately becomes the anterior surface of the neck. Thus, no vestigial enclosures could be associated with the precervical sinus.

In contrast to the theory of the branchial origin of lateral cysts and fistulae of the neck is the later concept which was brought out by Wenglowski,[8, 9] a Russian surgeon who published a monograph on this subject in 1913. Wenglowski spent five years investigating the origin of neck cysts and fistulae, studying serial sections of 65 embryos from 2.6 mm. to 40 mm., and dissecting 10 adult bodies. From this work, he made two important observations. The first was that in the human embryo the gill or branchial apparatus completely disappears by the end of the second month. This suggested to him the impossibility of that structure's having any part in the later developmental phenomena in the neck. The second observation was that the thymus gland anlage, from its point of origin as a diverticulum at the bottom of the third pharyngeal pouch, is transferred to the post-sternal space, leaving a duct or corridor in its path between the point of origin and the terminal point, where the gland subsequently reaches its full development. This change in position occurs as a result of the caudal movement of the heart in its pericardium simultaneously with the formation of the neck. Wenglowski argues strongly against the possibility that lateral cysts

of the neck have their origin from the branchial apparatus, first, because of the anatomic aspects and, second, because thus far in no higher vertebrate animal has any proof been given of the possibility of the growing of a ruptured gill fold into a fistula. He argues that the axis of the third gill arch is the external carotid artery, and in order for the second gill furrow to empty to the outside, it would be necessary for it to pass through the area between the internal and external carotid arteries. In reality, the fistulae do not reach this intermediate area, but lie in front close to the carotid sheath and extend from there toward the lateral pharyngeal wall. He argues, further, that since the branchial apparatus completely disappears by the end of the second month of embryonic life, it would be impossible for it to form the basis of fistulae below the hyoid bone except in the form of diverticula.

The course of the thymus corridor passes laterally from the pharyngeal wall and slightly downward to the area between the angle of the jaw and the lobe of the ear. Arching from here, it passes obliquely downward and forward medially, lying close to the lateral border of the thyroid gland. Its path is below the posterior belly of the digastric muscle, near its middle, and anterior to the carotid sheath, yet very close to that structure. It will be observed from this same picture that the lateral lobes of the thyroid gland have their origin in the floor of the fourth pharyngeal pouch and pass downward to join the median thyroid lobe. No cysts have been reported as having their origin from the lateral thyroid lobes. These observations on the embryo, however, must be regarded with caution, since there are many changes in the arrangement of the blood vessels in adult life. The thymus corridor usually shrinks entirely or in part. In exceptional cases, it persists either wholly or in sections. More often, it is the lower sections which persist. If the entire corridor persists, a complete fistula is formed, such as is shown in the case herein reported; if only a part of the corridor persists, an incomplete fistula will result. In this group lie those fistulae which open into the tonsil fossae and, after tonsillectomy, give the impression that the operation was incomplete.

The external opening of a lateral fistula is determined by a rupture of the fistula on the external surface of the neck, usually as a result of infection in the fistula, and a chronic discharge of mucous, serous or purulent material. The location of these openings along the anterior border of the sternomastoid muscle conform to a strict pattern and follow the course of the thymus corridor. Those fistulae which have been described as having their openings in the

larynx, trachea, esophagus and other unusual areas are no departures from the basic concept of their origin and development, but are examples of the variations in the clinical aspects resulting from infections of the original thymus duct.

In his studies of the 65 bodies of infants, Wenglowski found thymic anlage rests in serial sections 21 times, 12 times in the lower half of the neck, seven times in the upper half, and twice close to the pharynx. In the 10 adult bodies studied by dissection, he found these rests in two. Histologically, these rests are small canals or cysts, sometimes with squamous epithelium, sometimes ciliated epithelium, and at other times mixed epithelial lining. Hassall's bodies are sometimes found and the walls of the cysts are lymphoid in character, similar to the thymus. Further, he found in the fistulae walls several enclosures, such as transversely striped muscle fibers, cartilage, glands and phenomena similar to those found in the thymus duct wall. Particularly characteristic of lateral fistulae is the existence of lymphoid tissue in large quantities. In the walls of cysts formed in the upper part of the thymus duct may be found vestiges from the lateral pharyngeal wall which have been drawn into the tissue at the time of the descent of the thymic anlage. These vestiges may be glands, lymphatic tissue, cartilage and other elements. The lower one goes along the course of the duct, the fewer the occurrences of these vestiges. Round cell infiltration will be observed in the walls of those cysts and fistulae wherein infection has developed.

It seems to me that this concept of Wenglowski, built upon a long period of painstaking investigation both in the embryo and in adults, as well as upon clinical observation in his surgical practice, lifts the subject of lateral cysts and fistulae of the neck from a haze of mysticism, at least to a degree. To me, the pattern is much simpler than the fishgill theory. It permits one to approach surgery on these structures with greater assurance of a satisfactory outcome.

Within the scope of this paper are those cysts and fistulae having their point of egress about the pinna of the ear. These are of ectodermal origin at the caudal end of the first branchial cleft and are of little consequence surgically. Unless complicated by infection, they do not open inside the pharynx.

REPORT OF A CASE

B. A. C., white, female, seven years of age, gave a history of having since birth a discharging fistula, the outer opening of which was at the anterior border of the sternomastoid muscle near the

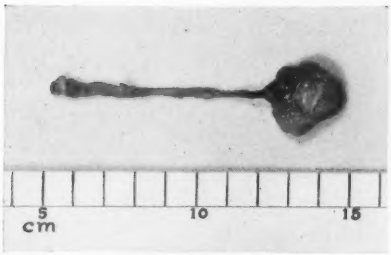

Fig. 3.—Tonsil and fistula intact.

sternal notch, on the right side. The fistula would heal and then break out, depending largely upon the presence or absence of colds and sore throat. She also had a history of recurring attacks of tonsillitis.

She was operated upon under general anesthesia. An elliptical incision was made around the external opening of the fistula and, by removing adherent tissue from the sac with scissors, it was followed in its upward course after piercing the platysma and superficial fascia. The ribbon muscles were pushed medially and, by extending the skin incision along the route, the lateral border of the thyroid gland was passed, the posterior belly of the digastric muscle came into view from above, and the duct seemed to arch medially toward the lateral pharyngeal wall. At this point methylene blue was injected into the outer end of the duct with a medicine dropper; on opening the mouth, it was observed that the methylene blue passed through and came out of the body of the tonsil on the same side at its upper pole. We had not intended to remove the tonsil at this operation, but now such a procedure seemed indicated. The tonsil was therefore dissected from above, according to our usual technique. It was observed that the fistula from the point of its emergence passed through the body of the tonsil and became connected with the tonsillar capsule, passing downward along the posterior surface of the tonsil to its lower pole. After reaching the lower pole of the tonsil, dissection was extended along the fistula until connection was made with our previous dissection from the outside. After completing the dissection, we were able to remove the tonsil and fistula intact through the mouth. The opening through the pharyngeal wall was closed below the tonsillar fossa by a purse-string suture

and drainage was provided externally after the skin incision had been closed.

SUMMARY

General agreement upon the origin of the thyroglossal duct has resulted in standardized criteria of diagnosis and treatment of cysts and fistulae having their origin from this duct.

The embryologic background of lateral cysts and fistulae of the neck influence the clinical, or anatomic and technical problems associated with them. Wide divergence of views in regard to their derivation has created much uncertainty and confusion as to their surgical management.

Early observers believed that lateral cysts and fistulae of the neck were derived from the branchial apparatus. In 1913, Wenglowski published a monograph disputing the possibility of the branchial origin of lateral cysts and fistulae, presenting evidence instead that these anomalies have their origin from persistence of parts or all of the thymus corridor.

A case is reported illustrating a complete lateral fistula of the neck having its internal opening in the right tonsil at the upper pole, and its external opening at the anterior border of the right sternomastoid muscle near the sternal notch. In this case, the tonsil was removed with the fistula intact. We were unable to find in the literature another case in which a similar procedure was used.

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XLIII

BRONCHOGRAPHY IN BRONCHIECTASIS IN CHILDREN

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The conquest of bronchiectasis by the surgeon has made bronchography of major importance. Bronchography in the adult patient is being achieved in many hospitals in a fairly satisfactory manner but in the bronchiectatic child it presents such great difficulties that the procedure is avoided as much as possible. Where it is being attempted the results are not wholly satisfactory.

Bronchiectasis can be cured by modern chest surgery. To effect a cure the thoracic surgeon has learned that he must excise every diseased portion of the bronchietatic lung.[1]

Kergin[2] reports that follow-up examinations of adults operated upon at the Toronto General Hospital over the past 19 years have abundantly demonstrated that bronchietasis is not progressive, that once cured the patient remains cured, and that the failures and imperfect results have been consequent upon incomplete excision of the diseased areas of the lung because of inadequate bronchography.

At the Hospital for Children, Toronto, since 1944, 28 consecutive lobectomies and five pneumonectomies have been performed[3] in children under 14 without a death and without an empyema and with no pleurobronchial fistulae.

Enough experience has been obtained on the surgical thoracic services to know that certain sequences of operative procedure greatly lessen the operative risk. For example, if the surgeon has successfully removed a severely bronchiectatic left lower lobe in the belief that the bronchiectasis was confined to that lobe, the slow development of unfavorable signs and symptoms may lead to the discovery of bronchiectasis in the lingula, the anlage of the middle lobe of the right side. The surgical removal of this lingula at a second operation on

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From the Department of Otolaryngology of the Faculty of Medicine, University of Toronto, and the Hospital for Sick Children, Toronto.

Read before the American Laryngological Association, St. Louis, Mo., April 24, 1947.

the left thorax is now much more difficult and much more hazardous than it would have been at the first operation.

Few thoracic surgeons believe that disease in such a lingula can be ascertained at operation by inspection or palpation. The presence or absence of bronchiectatic lesions in the bronchi of that lingula must be determined before operation by adequate lung mapping. Bronchograms, which four years ago would have been considered complete, are no longer considered adequate. The chest surgeon is now vitally aware of all the branches of the bronchial tree which are of importance. He is aware that these cannot be seen adequately in any one picture. Exposures in certain positions enable special bronchial branches to be seen and in those same positions other branches cannot be seen.

Therefore, today, before he will operate upon a bronchiectatic patient, the chest surgeon requires for study many bronchograms in many positions of all lobes of each lung. If he is not satisfied with the delineation of some part of the bronchial tree, he will demand more bronchograms of that particular portion, for he must plan his surgical attack and he cannot do that until the bronchography is complete to the last segmental bronchus.

In many articles published within the last 20 years and in some of the latest texts the demonstration of bronchiectatic lesions is dealt with as if it were almost contemptuously easy. For adults the catheter technique described by Jackson and Bonnier[4] is more or less followed in perhaps the majority of clinics. In many the procedure has long since passed from the laryngologist or bronchoscopist to the medical chest specialist and in many it is now passing from the latter to the radiologist. The factors determining this need not be discussed here. Suffice it to say that bronchography is of vital importance for the surgeon about to deal with a bronchiectatic lung. That complete adequate bronchography in the adult can be anything but easy is known only too well by those who endeavour to satisfy the thoracic surgeon of today.

Were bronchiectasis a disease only of adult life and did it not, in a high percentage of cases, have its beginnings in childhood, it would be necessary to add nothing more.

Unfortunately, however, bronchiectasis has been found present and of sufficient degree to require major chest surgery in very early years.[5] This fact is getting wider and wider recognition so that bronchography in children is becoming of great importance. In our

series of lobectomies four have been in children under three years of age. If the work is always arduous and frequently exasperatingly difficult in the adult patient, the difficulties of doing adequate bronchography in every bronchiectatic child are obviously greater.

Comparatively little has been written on bronchography in children. The article by Jackson and Bonnier[4] is followed by an excellent bibliography and recommends that the method used for adults be used with certain slight modifications for children. In spite of this recommendation bronchography in children has been used and is still being used far too little. Many hospitals rarely do a bronchographic study on a child and in very few has an attempt been made to meet adequately the needs of the children's medical chest service, for that requires work on a wholesale scale.

The reason is not far to seek. The catheter technique demands the cooperation of the patient, and while this can be obtained in selected, unusual children, often of the pay classes, it is almost so unobtainable in the very great percentage of miserable, coughing, apprehensive, unconditioned, little clinic patients as to make the catheter technique on the whole impractical for little children.

The authors advised against the use of the bronchoscope for bronchography in children for various reasons. They mentioned that too much of the lipiodol was coughed out and that often there was delay in the transportation of the patient to the x-ray department or in taking the pictures. We believe we have overcome those objections by performing the entire procedure on the fluoroscopic table in the x-ray department under general anesthetic.

Illustrations 3, 4 and 5 of the above article[4] excellently portray severe lower lobe bronchiectasis in the child, but there is no statement in that paper that the method can be used for all children, no matter how small, how sick, and how unconditioned, to map completely every portion of the bronchial tree to the satisfaction of the modern chest surgeon. And that is our present problem.

Amenable, cooperative children of 13 and 14 can eventually be completely mapped by the adult catheter technique. Such children may submit to several repeat bronchographies. A healthy, normal child of much younger age may submit to one bronchography but is likely to receive such an unfavorable impression that its cooperation for subsequent attempts is gone forever. Apprehension is ruinous. One cough at the wrong moment and almost instantaneously, before the radiologist can make his exposure, the iodized oil

has disseminated from the bronchial tree to the alveoli and the procedure has become a failure. Sedation does not safely overcome this factor.

Therefore, at the Hospital for Sick Children, Toronto, the practice described in 1942[6] has been continued. The bronchography is not preceded by sedation. It is performed under general anesthesia. The cough reflex starts working almost immediately after the operation is over and is a great factor in the invariably rapid postoperative recovery. No ill effects have resulted and the children invariably sit up to enjoy their evening meal. In 1942 we reported that general anesthesia in bronchoscopic work had been used 1268 times. Since then we have used general anesthesia in hundreds of cases for this purpose with excellent results.

During the past 12 months nearly all our bronchography has been completed at two delineations. Four members of the bronchoscopy staff take the work in turn.

The table top portrayed in that article has been abandoned. The present table has a space under it which permits the film to be taken in and out easily. The table is shockproof, completely grounded, and the air in the room is continually replenished so that there is no anesthetic risk.

The pictures, obtained by the method there described, have long since been considered inadequate because of the already mentioned increasing exactitude of delineation required by the surgical chest service. At that time, mapping of the upper lobes and the right middle lobe and the lingula was rarely demanded. Since then, there have been two instances of bronchiectasis confined to an upper lobe and many of bronchiectasis of the right middle lobe and of the lingula.

Consequently, every child, no matter how young, suspected of having bronchiectasis now gets complete mapping regardless of how many attempts may be necessary. A bronchograph should be made of only one lung at a time. For complete mapping of one lung the patient's body must be moved into various positions and roentgenograms must be taken in each of these. The time involved is of less importance than the efficiency and thoroughness with which the mapping is done.

However, this extra time gives rise to two new difficulties in the performance of bronchography with the bronchoscope with the patient under an anesthetic.

The first of these is the care that must be taken to do no harm with the rigid bronchoscope. This objection has proven more apparent than real. With the bronchoscope in the right or the left bronchial tree the patient's body can be turned to any desired position, even face down, without any trauma occurring. It is fair to state that this has been done many times without damage. However, it cannot be denied that these manipulations with the bronchoscope in situ necessitate great care and meticulous cooperation on the part of all members of the operating team and that acts against the popularity of the method.

The second difficulty is that of maintaining a long, quiet, even anesthesia. The anesthetic state is maintained by delivering ether, vaporized by an oxygen current, through a rubber tube attached to the anesthetic arm of the bronchoscope.

Anesthesia is not easily maintained through the bronchoscopes used, for the procedure, while similar to, is not identical with normal intratracheal anesthesia. In the latter a large, soft intratracheal catheter which tightly fills the larynx is used. In bronchography it has been our practice to use as small a bronchoscope as possible. In consequence, a greater ventilation of the bronchial tree occurs through the larynx outside the bronchoscope than occurs through the bronchoscope. There is also the additional handicap that the already small lumen of the bronchoscope is encroached on for quite a portion of the time by the suction tube or the tube for the iodized oil.

It is on the maintenance of a proper degree of anesthesia that the bronchography depends to a greater extent than on any other single factor.

To make the anesthesia more satisfactory we are now using thin-walled metal intubation tubes. The advantages and technique are described later. We believe that this alteration in method has greatly diminished the difficulties of bronchography in bronchiectatic children.

It is no longer considered sufficient to demonstrate the presence or absence of bronchiectasis in each of the five pulmonary lobes. Clinical experience is demonstrating repeatedly that lobes infrequently are completely diseased, but rather that the bronchiectasis involves certain segments of lobes leaving other segments healthy. The result is that an entire lobe is no longer considered the surgical unit. The bronchopulmonary segment has become the surgical unit and in the adult is now lending itself to satisfactory removal.

Radiological Report on Bronchogram No.....

Patient Ward Date

Surgeon Anesthetist

LUNG	LOBE	SEGMENTAL BRONCHI		ABNORMAL CYL.	ABNORMAL SAC.	SHOWN IN FILM NO.
Left	Upper	Upper Apical posterior	1 & 2			
		Anterior	3			
	Lingula	Superior	4			
		Inferior	5			
	Lower	Superior	6			
		Anterior medial	7 & 8			
		Lateral	9			
		Posterior	10			
Right	Upper	Apical	1			
		Posterior	2			
		Anterior	3			
	Middle	Lateral	4			
		Medial	5			
	Lower	Superior	6			
		Medial	7			
		Anterior	8			
		Lateral	9			
		Posterior	10			

Notes:

Signed.....

Radiologist.

Fig. 1.

REQUEST FOR BRONCHOGRAPHY Date.....

Patient Age..... Ward.....

Segmental bronchi to be outlined: .....

.....

.....

.....

.....

Signed..... .....

Fig. 2.

While it is possible that the excision of segments may become a recognized procedure for children, no patients have yet been operated upon in this fashion (except the excision of the lingular process) at our hospital. It has been considered in several cases but in each instance the final decision has been for lobectomy and subsequent examination of the excised lobe has proven the decision wise.

Jackson and Huber[7] in 1943 proposed a nomenclature of the segments of the bronchial tree which has met wide approval and use. It has been adopted at our hospital until such time as thoracic surgeons adopt a standardized nomenclature. Their scheme was accompanied by an excellent colored chart which, however, had the disadvantage of being difficult to reproduce, so we have been quick to adopt the numbering of the bronchial segments proposed by Overholt and Langer.[8]

Their modification has been followed in the printed form used by our radiologist to report on the films of each bronchography (Fig. 1). It can be seen that this form makes it easy for the surgeon and the medical chief to agree readily on precisely what bronchopulmonary segments require delineation at the next bronchography. Their request comes to the bronchoscopist on this form and greatly facilitates his work (Fig. 2).

Following is a brief outline of our technique.

1. Induction of anesthesia.

2. Suction aspiration of the bronchial tree through a bronchoscope.

3. Insertion of special intubation tubes.

4. Insertion of a small catheter into the bronchial tree.

5. Injection of iodized oil.

6. Spot films (Bucky diaphragm not used).

7. Large-sized regular films (Bucky diaphragm used).

The whole procedure takes place in the x-ray department.

The patient comes without sedation. The success of the procedure depends, in part, on the rapid return of the cough reflex once the anesthetic has been stopped. No local anesthetic is used.

The excitement of coming for operation excites coughing and this coughing is encouraged in order to free the bronchial tree of as much of the purulent material as possible.

We are in agreement with Smyth and Schall[9] that thorough cleansing of pus from the bronchial tree is essential to good mapping because iodized oil cannot fill bronchi plugged with heavy bronchiectatic secretion.

The conscious patient lies on the fluoroscope table top on his side with the more diseased lung dependent and with his shoulders perpendicular to the plane of the table. This position is of assistance to the anesthetist during the induction of anesthesia. Induction is carried out as rapidly as possible in order that the cough reflex may not be stimulated and result in the expression of material from the lung periphery into the main bronchial tree which would interfere with aeration. The induction is continued until a deep enough anesthesia is reached to allow the introduction of the bronchoscope without any damage to the patient or embarrassment to the bronchoscopist and without arousing any reflex from the introduction. Then the patient is turned on his back and a 5 mm. or a 4 mm. bronchoscope inserted.

Suction is used through the bronchoscope to clear the trachea and the main bronchi. The anesthesia is diminished during this procedure, so that deeper respiration and cough may favor the expulsion of exudate from the inaccessible parts of the bronchial tree.

When no more material is being produced for suction, the anesthetist increases the depth of anesthesia to the degree he considers

advisable for bronchography. The bronchoscope is withdrawn and a special intubation tube of as large a size as possible is inserted into the larynx through the long slit on the side of an anesthetist's laryngoscope or through a Jackson laryngoscope from which the slide has been removed. Special metal introducers devised by our chief anesthetist make the insertion easy and safe. These tubes are very thin-walled and are really very short intratracheal tubes with a large lumen; they should not be inserted without the introducer. Care should be taken to select a tube of suitable diameter in order to avoid trauma to the larynx. Through this anesthesia is easily maintained by an ether hook in the corner of the patient's mouth.

If a Jackson laryngoscope is used, an ordinary, soft rubber catheter which has been slightly shortened by cutting off the olive tip and the side apertures is passed by the bronchoscopist through this intubation tube and by fluoroscopic guidance is inserted into whatever part of the bronchial tree the radiologist desires. The bronchoscopist anchors the tube with his fingers.

Maintenance of anesthesia now becomes relatively simple. The patient's body can be turned into any position desired without any danger of trauma. The two difficulties previously mentioned have been surmounted.

Burrell's syringe, partly filled with cold iodized oil, is fitted into the end of the catheter by the bronchoscopist's assistant. The oil is injected very slowly, as the radiologist desires.

For bronchography for the right lung assistants turn the patient's body to the left anterior oblique chest position and use sand bags to maintain the position. A nurse at the foot of the table holds the patient's ankles, or they can be secured by orthopedic anklets and ties.

The oblique positions are desirable because the bronchial tree divides in a direction oblique to the horizontal (when the patient is in the horizontal position). In consequence, the oblique positions permit clear separation and identification of the secondary bronchial divisions.[10]

With the patient in this oblique position the table is tilted head down to approximately 20° from the horizontal. The injection of iodized oil is begun and the upper lobe bronchi are filled until well delineated. A "spot" film is taken at the desired moment (before alveolar flooding has taken place). The table and the patient are then maneuvered into the second position. The table is tilted head

up to approximately 10° above the horizontal. This facilitates filling the lower bronchi and the middle lobe of the right lung. A second "spot" film is taken after optimum filling has taken place.

Following this the patient is quickly returned to the horizontal and three films are taken with the Bucky diaphragm in the oblique, anteroposterior, and lateral positions.

The radiological data are:

	M.A.	K.V.	T.S.D. TIME 48
3-year-old child			
Oblique	150	74	1/20
A.P.	150	70	1/20
Lateral	150	50	1/20
10-year-old child			
Oblique	150	76	1/10
A.P.	150	72	1/10
Lateral	150	80	1/10
		Bucky	1/10

If the middle lobe bronchi have not filled properly, the patient is turned face downward and the bronchial filling watched in the fluoroscope until the desired picture can be taken.

The oil is drawn into the bronchi by inspiration; it is never forced in by the syringe.

The keynote to good results lies in the absolute cooperation of bronchoscopist, anesthetist and radiologist. All the pictures must be taken as rapidly as possible.

At the conclusion of the procedure the intubation tube is pulled out. The patient comes rapidly out of the anesthetic and it is rare that there is any embarrassment to those watching the postanesthetic recovery. The work is performed in the morning between 10:00 and 12:30 and the child is usually sitting up at 4:30 p. m. enjoying supper, and almost invariably is discharged the next morning. Bronchography of the left lung is done on a subsequent occasion after an interval of several weeks. The main consideration is that most of the iodized oil injected at the first bronchography should have vanished so that the delineation of the left bronchial segments is not impaired by overlying iodized oil in parts of the right lung. The patient's body is first placed in the right anterior oblique position. The face-down position is sometimes necessary for proper mapping of the bronchus leading to the lingula.

In the past the main problem has been the proper delineation of the bronchi to the right middle lobe, the lingula, and the right and left upper lobes. The delineation of all of these is very satisfactorily accomplished by our present technique. The filling of the posterior segment of the left lower lobe is sometimes difficult.

Several modifications of the technique have been discarded. Among them are the following.

1. The Thomson catheter[11] was given an adequate trial. (It was inserted without the use of the rustless wire carrier.) It has only one opening—at the distal end—and this must serve to convey oxygenated ether and later iodized oil. This small end aperture will only maintain anesthesia by the use of pressure and then only for a short time, and during that time the ventilation of the patient is precarious to say the least. Iodized oil is then quickly substituted for the oxygenated ether and during the period of injection there is no ventilation. As soon as the oil has been delivered, the catheter must quickly be pulled back into the trachea so that the anesthetist may blow in oxygen. Thus the concern of the anesthetist and the bronchoscopist is with the patient and not with the bronchography and the radiologist is lucky if his pictures are good.

Cutting holes in the sides of the catheter helps the anesthetist's problem but the consequent escape of the iodized oil ruins the radiologist's efforts.

2. The passage of a large intratracheal catheter and the insertion through this of a fine soft catheter has also been tried.[12] The method is to insert as large an intratracheal catheter as possible into the trachea proximal to the carina and to insert a glass Y tube into its proximal end. Through one arm of the Y the anesthesia is delivered and through the other arm a long fine soft catheter is inserted, the end of which is directed by gravity and fluoroscopic guidance to any desired position in the bronchial tree. The iodized oil is delivered through it by the syringe already mentioned.

This method is not as easy as it sounds. The main difficulty is the maintenance of the anesthesia, for a catheter inside the intratracheal catheter makes for greater resistance to ventilation. The method described in this paper is very much easier.

3. Dr. Basil Bradley, one of my colleagues, is developing a modification of a two-way irrigating urethral catheter. Very satisfactory bronchograms have been produced with its aid. Its future depends upon the ability of the manufacturer to produce catheters which will stand the wear and tear of hospital use.

CONCLUSION

Many years ago Kernan[13] stated, "For the purpose of lung mapping the right amount of lipiodol should be injected into the lung and it should be put in the right place to fulfill the requirements. It is necessary to introduce it under the guidance of the eye. The use of the fluoroscope offers further guidance." The problem could hardly have been presented in fewer words. The intervening years have shown that the procedure is not as easy as it sounds but we believe we have made a further contribution to the solution of a difficult problem.

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XLIV

CHANGING CONCEPTS OF THERAPY OF CHRONIC SINUSITIS

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I wish to limit this discussion primarily to certain intumescent states, of functional type, which complicate the treatment of chronic suppurative sinusitis. Most forms of rhinitis are now referred to as synonymous with sinusitis. These are mucous membrane reactions. As such they develop hypoactive and hyperactive rates of mucus production. The considerations which follow deal with hyperactive states of chronic character. They play an important part in the production of chronic suppurative sinus disease. An understanding of them is necessary for successful therapy. Principles of surgical relief have been adequately presented in recent publications.

The paranasal sinus mucous membranes are extended portions of the nasal mucous membrane. Pathologic changes in the nasal mucous membrane are accompanied by similar pathologic changes in the sinus membranes. These changes may be of infectious origin resulting in nonsuppurative or suppurative rhinosinusitis.

They may, however, be abnormal tissue reactions resulting from systemic disturbances or long exposure to local irritants. There must be recognized, therefore, a nonsuppurative type of chronic rhinosinusitis, having as its chief characteristic the uniform involvement of all the nasal and sinus membranes. True localized chronic sinusitis, usually suppurative, is recognized when there is greater pathologic change present in one or more paranasal sinuses than exists in the general expanse of nasal membrane. It is chronic when it is not within a reasonable time self-terminating and is to a certain extent self-perpetuating. Successful therapy is entirely dependent upon correction of the self-perpetuating factors which are the basis of chronicity. Changing concepts of therapy have resulted from:

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1. A broader understanding of the factors producing functional types of rhinitis.

2. Continued contributions to our knowledge of nasal physiology.[1, 8]

3. Certain practical applications of endocrinology to rhinology.

4. Further successful clinical experiences with nonseasonal allergic rhinitis.

5. Excellent studies in surgical anatomy and histopathology which have helped to make surgical treatment more effective.

Functional changes affect only the mucous membranes. Discomfort is complained of only when the membrane is producing too much or too little moisture. Patients become conscious of discharge in the nasopharynx when mucus is abnormally fluid, excessive, viscous, mechanically, chemically or biochemically irritating, odorous, or obstructive. Some of the dry states are of interest. Certain drugs, notably belladonna, reduce humidification and ciliary activity. Certain psychic causes reduce the function of the nasal glands, producing dryness of the nose and throat, such as that experienced in public-speaking by those unaccustomed to it. Function is reduced in certain febrile diseases, such as influenza and typhoid. There is a hypoactivity in debilitated persons, in those leading sedentary lives and in cardionephritics. Typical atrophic rhinitis and atrophy in the aged are other examples. In this discussion, however, we are more concerned with the hyperactive states and their effect in the treatment of localized chronic suppurative sinusitis.

Intumescent rhinitis might frequently, but not exclusively, be described as a metabolic turgescence of the nasal and sinus membranes. It is characterized by a smooth, engorged, deeply colored membrane in which blood and lymph vessels are relaxed and overfilled. It is not allergic in origin and is different from allergic rhinitis in its histopathology. The clinical findings in the sinuses are not presumptive but may readily be demonstrated by roentgen studies with film exposure light enough to retain soft tissue outline or by the more tedious method of filling the restricted sinus cavities with radiopaque oils. The presence of the chronic intumescent state usually constitutes a symptom of some systemic disturbance. It may result from long exposure to irritating atmospheres.

The management of this troublesome and persistent form of nasal obstruction is concerned primarily with functional and not

structural correction. It is to be distinguished from hypertrophic rhinitis which is characterized by an actual tissue increase and as such is an irreversible pathologic state. The intumescent state has a pronounced retarding effect on drainage and ventilation. It usually can be corrected. Its continued existence should not long be allowed if the patient is cooperative and diagnostic and therapeutic facilities are available. Local therapy is of little value.

The condition occurs most frequently with hypothyroid states, with excess weight, low metabolic rate, and low afternoon temperatures; in patients who eat starchy foods to excess or drink, particularly beer, excessively and become overweight. It occurs with certain other endocrine disturbances, and in patients whose air tracts are long exposed to irritating atmospheres. "Colds" that precede menses are related to it. Nasal obstruction of pregnancy may be particularly troublesome and persist through the entire period of gestation. It is not infrequently, in the later stages, accompanied by chronic suppurative sinusitis. One patient was reported[9] whose long persistent and severe nasal obstruction disappeared completely after an oophorectomy and recurred each time ovarian therapy was instituted.

Observations have been made of a definite food-sensitivity reaction with smooth, red, intumescent membrane, observed to be due to low tolerance to sugars, starch, or alcohol. Nash[9] reported a long-suffering young patient who had many nasal operations, therapeutic procedures and climate changes whose intumescence was cleared by the simple expedient of removing candy.

Excessive and prolonged nasal medication may produce an intumescent state. Preservatives in solutions prepared for use as nose drops such as phenol, menthol, eucalyptol and especially chlorobutanol may have to be eliminated. The use of vasoconstricting solutions should be held to a minimum. One of the most efficient of these is privine hydrochloride. If it is prescribed, its use should be held to a minimum and a "no refill" order given. We have observed that this preparation in certain individuals actually produces a high-grade persistent intumescence. Four to five days' complete privation from the solution eliminates this reaction.

Physical factors such as low humidity, thermal changes for individuals particularly susceptible to winter inside-outside temperature variations, and summer air-conditioning are to be considered. Garage workers who are exposed to irritating gases, people who work in exceptionally dusty atmospheres, musicians who work in

close smoky atmospheres in road houses and night clubs are frequently affected. Psychic factors are occasionally of great importance, for not infrequently headaches and intumescent states disappear after an emotional crisis has been dissipated.

The problem of intumescence is fundamentally one of systemic disturbance. It requires careful checking of bodily functions and habits of eating and drinking as well as a study of the patient's home and working environment.[10] The aid of an understanding internist can be of great value. A large proportion of these patients respond to a weight-reducing program and elevation of a depressed metabolic rate, with thyroid therapy regulated to requirements. The adult patient is usually given one grain of thyroid daily the first week, two grains daily the second week, and three grains daily the third week. Why do we consider a form of nonsuppurative rhinosinus reaction in a study of chronic sinusitis? Because an intumescent membrane obstructs ventilation and drainage and encourages recurrence of suppurative sinus disease. Electrocoagulation or surgical sacrifice of turbinate tissue for this condition leads to irreparable damage of the automatic air-conditioning mechanism of the nose. The lower turbinate governs the physiological adjustments of the most spacious part of the nose. The middle turbinate occupies the position of and regulates the greatest variety of nasal functions. The cleansing-filtering process accomplished by a rapidly moving mucous adhesive layer is a function of the cilia and is completely dependent on moisture. Turbinate injury by surgery, diathermy or cautery disturbs this element of nasal physiology and should be reserved for hypertrophic states and organic changes. The rule is to reserve operative procedures for organic disease and to avoid operations in functional disorders. In London in 1596 Thomas Cogan[8] wrote "There is not any one more annoyance to the health of men's bodies in this Realme of England than distillations from the head, commonly called rumes, the occasion of which some impute to much drinking of Beere, but I think the great moisture of the air of this Realme, for we have a rainy and cloudy skie. . . . " This is not a problem local to the "Realme of England," however, because a recently returned Rhodes scholar commented, "I am astounded to observe that Americans are always spitting."

The allergic type of chronic rhinosinusitis is currently the most challenging problem. The allergic membrane is an irritated membrane but is pale because the capillaries permit the escape of ameboid white cells and fluid but no red cells. Sinuses cannot be cleared satisfactorily if there is coexistent allergy which persistently obstructs

ventilation and drainage. The allergic mucosa responds poorly to even mild infections. With coexistent allergy and chronic sinus disease, the best therapeutic results follow when the infection and the allergy are treated simultaneously. Nonseasonal allergic manifestations are of most interest to rhinologists. It is necessary to meet nonseasonal allergy problems because of the frequency with which patients having this condition present themselves for assistance. Skin testing, as frequently practiced, is ineffective in leading to the patient's relief from the rhinologist's point of view for two reasons:

1. A general laboratory technician makes and reads the tests and there is a consequent loss of a certain clinical perspective in a multiplicity of tests, especially if there is a large number of positive reactions.

2. A trained allergist makes the tests and establishes a general routine for allergic gastro-enteritis or dermatitis. This results in too little attention being given to the inhalant group which is of more interest to the rhinologist treating the air tract.

It is preferable, therefore, to have the inhalant tests for the allergens which most affect the air tract done and observed in the rhinologist's office or clinic. Here the tests can be clinically appraised and the advisability of type and intensity of therapy determined. Usually the offending group and the one most promising from the standpoint of treatment will be found simply and quickly by testing for the common inhalants. Chief of these is the epidermal group which includes substances growing on skin such as wool, feathers, horse and cattle hair, hair of pets, particularly dogs and cats, and particularly house dust. Molds, orris root, and other allergens may be included according to indications and the personal clinical experience of the examiner. It is well to remember that, in general, too much dilution brought about through administering too many substances in combination reduces the effectiveness of desensitization therapy. Testing is reduced to a practical and less time-consuming basis.

Desensitization therapy against the inhalants is highly successful. It is important to measure carefully graduated doses, refrigerate diluted solutions and seal needle punctures to insure that carefully prepared and measured material is absorbed by the patient in the amount intended. Complete therapeutic details have been published by Hansel,[3] Shambaugh,[2] Huber[4] and others. A trained assistant can take over this valuable but somewhat time-consuming

adjunct in the therapy of a troublesome type of chronic rhinosinusitis. If the patient exhibits any of the features described under intumescent rhinitis all forms of therapy are combined.

Our first patient to be put on desensitization therapy was an 84-year-old, still vigorous male patient who demanded that something be done about his sensitized nose and sinuses. His sinus washings were clear, his skin tests positive, eosinophiles were present and all tests pointed to a perfect trial. Since we had little confidence in the method, his complete relief was an entire surprise. Since that time approximately 200 patients have been treated by house dust desensitization with results that are undeniably good. We have noted that a maintenance dose, a repetition of the top dose, is desirable at intervals varying from four to six or eight weeks. With careful selection of patients the number of frank disappointments will be small. This additional form of therapy is becoming recognized by some departments of otolaryngology which have, or are planning, sections where expert observation and therapy will undoubtedly further improve results in nonseasonal allergic rhinitis.

Because our knowledge is still limited some patients will resist all constructive therapy. Three types of less conservative local therapy are mentioned because allergic nasal conditions can be difficult problems.

1. The galvanic current can be applied to an electrolite in the nose. While not to be too much encouraged, this method has produced satisfactory relief in many instances.

2. The bipolar nasal electrode, using electrocoagulating current, may be applied to all redundant tissue of the inferior turbinate. It is effective and easy to use.

3. Sclerosing solutions injected under the mucosa give satisfactory shrinking. Five per cent sodium morrhuate or sylnasol* give relief for a time varying from a few months to many months.

The more we learn of proper testing and desensitization and of the nasal reactions to certain systemic derangements, the less these irreparably damaging local measures will be used. Certainly one must understand the known facts regarding the origin and therapy of the common types of chronic rhinitis before great success can be achieved in treating individual chronically diseased sinuses.

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*Five per cent solution of the sodium salts of certain fatty acids from oil extracted from the seeds of the psyllium group.

I would like to comment briefly on some phases of treatment of chronic suppurative maxillary sinusitis. Early treatment prevents contamination of other sinuses. Sound knowledge of regional anatomy, physiology and histopathology are essential to produce good results.

Antrum infections of dental origin are more destructive than other types. They invade the normal defensive mechanisms of the mucous membrane by first invading the subepithelial stroma.[7] In my series 11 per cent of the chronic suppurative infections were caused by contamination from diseased tooth roots. A considerable proportion of the remaining 89 per cent were residual infections from virus diseases, chiefly influenza, which have a highly neurotropic toxin with a particularly paralyzing effect on cilia. The treatment is apparent and consists of irrigations which dilute the toxins to the point where normal ciliary activity is restored. For this purpose I use warm Ringer's solution* or Fisher-Bledsoe solution.[11]** The latter is particularly useful because it is easy to store as a concentrate. Twenty-two cc. of concentrate are diluted to 500 cc. with sterile water and used at body temperature. I have not been able to demonstrate an advantage in the local use of penicillin or sulfonamide solutions for irrigation in the treatment of chronic suppurative maxillary sinusitis.

I have no fixed practice regarding the use of the cannula through the natural opening or puncture of the inferior meatus. A wide variation in the anatomy requires discriminating application of principles to the case in hand. It is usually easy to judge whether the patient's osseous structure is light or heavy. If light, the lower meatus approach is simple, painless and leaves the natural opening unimpeded for return flow. Ciliary streaming is toward and through the ostium. Proetz[1] has emphasized that this is in itself a functioning organ and injury should be avoided wherever possible. Van Alyea[5] prefers the middle meatus approach to minimize the patient's discomfort. A search is made for an accessory opening, then for the maxillary ostium. If no opening is readily found a sharp-pointed cannula is pushed through the membranous portion of the naso-

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*1000 cc. Ringer's solution contains:

Sodium Chloride	7.0
Potassium Chloride	.3
Calcium Chloride	.25

**1000 cc. Fisher-Bledsoe solution contains:

Sodium Chloride	263.7
Dried Calcium Chloride	21.0
Potassium Chloride	10.6

antral wall just above the inferior turbinate slightly posterior to the midline. He carefully guards his clinical routine, however, by stating that when prolonged search would produce damage that would offset the benefits derived from sinus lavage, the lower meatus approach is substituted. Great credit is due those who have painstakingly demonstrated the surgical anatomy involved in the surgical treatment of chronic suppurative sinusitis. The dictum that has resulted is: "Restoration and maintenance of adequate drainage channels is to be done with the least possible damage to functioning nasal and sinus mucosa and bone structure."

The chronically infected antrum which requires a double needle for irrigation or which shows no sign of healing after a reasonable number of well-spaced irrigations is in need of ventilation and requires a fenestra. To insure complete success the window should be as large as the anatomy of the lower meatus safely permits. The edges should be thoroughly smoothed with a fine rasp. Membrane fragments, bone chips and bone dust must be thoroughly irrigated away. No flaps are left or needed. When sinuses are opened and left exposed to blasts of dry inspired air the sinus membrane, being sparcely supplied with glands, undergoes metaplasia, becomes painful and accumulates sticky secretions and crusts. The antrum window is successful because it lies away from the ostium and is protected from incoming air blasts by an unimpaired lower turbinate. The Caldwell-Luc operation is reserved for tumors of the antrum, dental cysts in the base, osteomyelitis, and certain traumas where direct inspection is imperative.

CONCLUSIONS

Successful therapy of chronic suppurative sinusitis is entirely dependent upon knowledge of how to correct factors which produce chronicity.

Changing concepts of therapy have resulted from several fields of research:

1. Practical applications of endocrinology to rhinology.
2. Continued contributions on nasal physiology.
3. Better understanding of functional types of rhinitis.
4. Successful clinical experiences with nonseasonal allergic rhinitis.
5. Contributions of surgical anatomy.

Surgical procedures should be avoided for functional disorders and reserved for organic disorders.

Rhinologists are best prepared to give relief to patients with allergic rhinitis of nonseasonal character and should assume that responsibility.

For surgical correction of chronic suppurative sinusitis all fields of research now direct that, "Restoration and maintenance of adequate drainage channels is to be done with the least possible damage to functioning nasal and sinus mucosa and bone structure."

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XLV

EAR NOSE AND THROAT SURVEY OF CHILDREN IN THE SCHOOL FOR THE DEAF AT JACKSONVILLE, ILLINOIS

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CHICAGO, ILL.

AND

ROBERT SPEAS, M.D.

TERRE HAUTE, IND.

This survey was planned so that it would be of interest and potential benefit to both the Jacksonville School and the Illinois Eye and Ear Infirmary. The fact that both are state institutions facilitated the survey of the students and the classification of their types of deafness, through the teacher and the physician working together.

It had certain objectives to fulfill:

1. To examine each student, using the hearing and tuning fork tests, audiometric examinations, and labyrinthine tests, and to attach to each case a definite and accepted medical diagnosis. If the case was found to be a type of deafness associated with an ear infection, there was the possibility of individualized medical and surgical treatment in addition to speech training. If the case was found to be due to congenital causes, it was planned to determine the extent of residual hearing and to suggest an individualized auditory and speech training program.

2. To find the etiological factor of each case and to determine whether this factor and the degree of deafness had any correlation with the child's behavior in school and every-day life.

3. To provide the school's educational staff with medical terminology and the classification of deafness.

This survey was made possible through the cooperation of General H. E. Thornton of the State Department of Public Welfare and Mr. Daniel T. Cloud, Superintendent of the School for the Deaf in Jacksonville, Illinois.

Presented before the Chicago Laryngological and Otological Society, December 2, 1946.

4. To classify and grade each case so that future plans for school and job-training programs could be accomplished for each student individually.

5. To classify the causes of deafness; to trace any hereditary traits or congenital factors which may be dominant or recessive; and thus to be able to advise each student, individually, as to future plans for marriage and parenthood.

Children enter the school for the deaf in Jacksonville very early in life. The youngest subject we examined was four years old; the oldest 21. According to the school records there were 15 instances of brothers and sisters attending. The pupils remain in the institution during the school year until they complete grade and high school courses. The school offers very satisfactory facilities for hospitalization and isolation of sick children and a well trained nursing staff.

Studying the charts of the students we found complete descriptions of the physical development and the condition of the children, the course of diseases occurring during the school year, and the birth data. Otological reports were added only in cases which needed special otological intervention. Examinations of the ears, nose and throat, and hearing and vestibular tests had not been noted.

In the course of our study we attempted to evaluate the history of each case as given to the school physician as compared with the clinical findings. It is a very common occurrence to find that the parents of a hard-of-hearing child assume that the difficulty came from some illness or accident in early childhood, even when there is a definite family history of deafness or defective hearing. In most instances there was no evidence that the child had ever had any functional hearing.

We had no opportunity to speak to the children's parents but we learned that during the past year the social worker had interviewed each new applicant for the school and had questioned the parents to great length to find indications of hereditary deafness.

It is understood that there are many difficulties in obtaining accurate family histories which requires the cooperation of parents and relatives of the deaf child, but we feel that medical records, particularly those of the deaf, should include complete data on inheritance of the deafness for as many generations back as they can possibly be obtained. It is also of importance to include in the record data concerning the incidence of illegitimacy and the pregnancy histories of the mothers.

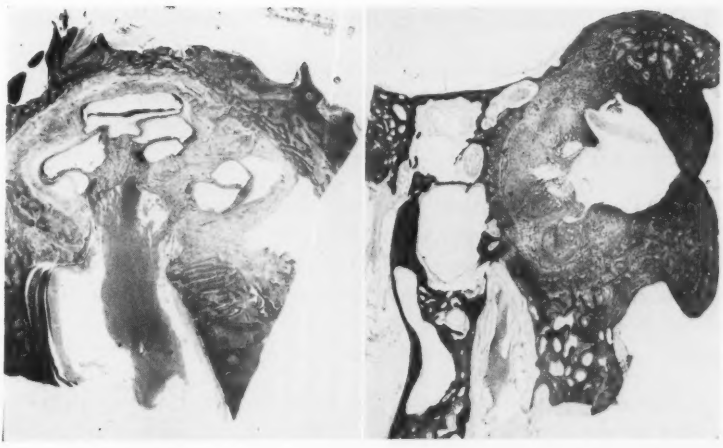

Fig. 1.—Vertical section through the cochlea and the inner canal of a congenitally deaf child. The acoustic canal is enlarged. The eighth nerve is present and partly atrophic. The modiolus is deformed and not completely ossified. Rosenthal's ganglion is atrophic. The organ of Corti is partly undeveloped, partly deformed and attached to Reissner's membrane. The vascular stria is flattened and degenerated.

Fig. 2.—Vertical section through the cochlea of an adult man with postmeningeal deafness. The inner canal is narrowed, its lumen with the exception of the seventh nerve, empty. The spiral ganglion in the modiolus is absent. The scalae are filled with a rather compact bone with a tendency to produce haversian systems. The geniculate ganglion, the tensor tympani and the bony eustachian tube are normal.

The Conference of Executives of American Schools for the Deaf has given a new definition and nomenclature, which classification has recently been approved by the American Otological Society. It is as follows:

1. The Deaf: Those in whom the sense of hearing is nonfunctional for the ordinary purposes of life, divided into distinct classes based on the time of the loss of hearing.

(a) The congenitally deaf.

(b) The adventitously deaf—those who were born with normal hearing but in whom the sense of hearing became nonfunctional later through illness or accident.

2. The Hard of Hearing: Those in whom the sense of hearing, although deficient, is functional with or without a hearing aid.

We would like to present some illustrations of the main types of deafness comparing clinical with anatomical findings. Three prominent types were established histologically and are approved in the otological literature.

The first type described by Mondini and named after the author shows a nearly normal pars superior of the labyrinth but a marked dilatation of the vestibular aqueduct. There is a defect of the upper part of the modiolus and consequently a common hole towards the cupola known as the "scala communis". Corti's organ is partly missing and partly imperfectly developed (Fig. 1).

The second type represents the group in which the changes are limited to the membranous parts. Here one can find proliferation of the epithelium of the vascular stria which appears in some sections as a tumor-like mass. The stria itself may be defective and the blood vessels obliterated or not developed.

Corti's organ is displaced or entirely missing. The pars superior of the labyrinth may show slight degenerative changes in the macula and the crista.

The third type is associated with complete aplasia of the labyrinth in the presence of the external and the middle ear. This form is very rare.

Deafness through inflammation, whether of tympanic origin or caused by epidemic meningitis, is always characterized by new tissue formation in all spaces of the cochlea and the vestibulum. The filling tissue, fibrous, cartilaginous, or osseous, almost completely obliterates the scala, the vestibule, and the semicircular canal (Fig. 2).

There have been cases of congenital deafness reported in which the entire cochlea and vestibulum were normal. In these cases one has to assume a central lesion in the acoustic nuclei and in the auditory cerebral pathways.

We preferred, in our study, the clinical classification based on hearing tests and vestibular stimulations. Considering the fact that the available histories were inadequate in regard to hereditary data, we have used the findings in the ear in connection with the labyrinthine tests to determine whether deafness was congenital or acquired.

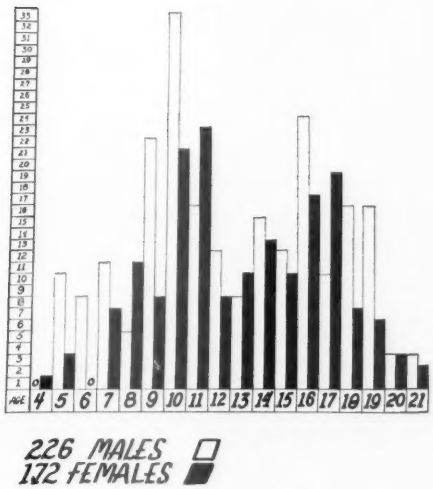

Fig. 3.—Distribution of male and female deaf according to age.

In the acquired form of deafness we had to differentiate deafness from meningitis, and from other causes, such as otitis or labyrinthitis. This classification appears more practical from the medical point of view. Congenital deafness, according to this division, is associated with a functioning vestibular apparatus and occasionally with some hearing residuals. Postmeningeal and postlabyrinthitic deafness are characterized by a complete cessation of all functions.

The Rinne and Schwabach tests were made with a G-1 fork (317 double vibrations). The C-64 and C-2048 forks were used and the number of seconds they were heard by the examiner after the tested ear failed to hear was noted as a minus so-many-seconds from the tested ear. As part of the hearing test a harmonica was used. Whispered and spoken voice were examined separately in each ear. Bárány's noise apparatus was used as a masking device.

Turning tests were performed on every child according to the classical procedure. Whenever a negative turning reaction was encountered, a caloric test was done. This test was made on only about one out of every five patients, and in every instance with the same results as with turning. Nystagmus was noted as 1, 2, or 3 degree.

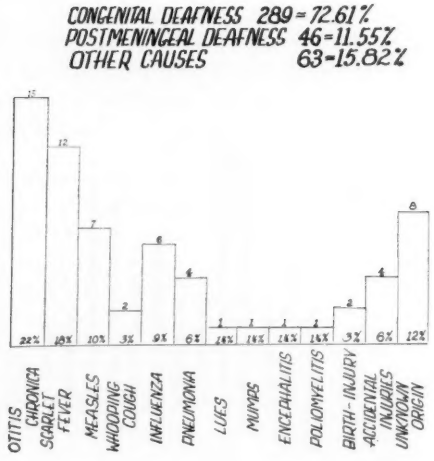

Fig. 4.—Causes of deafness in Jacksonville School 1946.

Three hundred and ninety-eight children were examined. Of these, 172 were females and 226 were males. They are listed in Fig. 3 as to age and sex.

It was very difficult to obtain an accurate history from the pupils. In most instances we had to rely on the history as given by the parents and noted in the child's school record. Previous to last year the records are not complete and are difficult to evaluate. The following figures were arrived at from the history and the findings, and are as accurate as we could possibly make them. This chart shows the incidence of congenital deafness and congenital hard of hearing and postmeningitic deafness. The first two groups were considered together. The deaf and hard of hearing from other causes will be considered separately.

The incidence of deafness is shown in Fig. 4.

Table 1 presents information concerning the appearance of the ears of these children. In all the children examined the auricles and the external canals appeared normal and without signs of malformation or stigmata of degeneration. As might be anticipated in a group such as this, there was practically no correlation between the appearance

TABLE 1.

APPEARANCE OF DRUM MEMBRANES

	CHILDREN	PER CENT
1.) Bilateral normal appearing drum membranes	280	70.35
2.) Atrophic drums with a blush of the promontorium	10	2.5
3.) Marked retracted drum membranes bilaterally	33	8.2
4.) Bilateral adhesions in the membranes	60	15.0
5.) Chronic suppuration with large destruction	15	3.75

of the membrana tympani and the degree of hearing present. There was one exception. Practically all of the children with chronic otitis media had hearing that could be utilized in the class room. Again we would like to call attention to the small percentage of pupils who were hard of hearing because of chronic ear infections. In years past, the percentage due to this cause was much higher.

There was a definite blush of the promontory wall with intact membrana tympani in 10 children. The drum membrane appeared somewhat atrophic but otherwise normal. This was observed only in children considered congenitally deaf. These children could hear some sounds but did not have sufficient hearing for practical use, as was shown by spoken voice or audiometer tests.

Hearing. At this school a trained audiometrist does the audiometric examinations. The tests appeared to be performed accurately. It is routine to test every child at least once during each school year and more frequently where there seems to be improvement or progressive hearing loss. Generally speaking, the audiograms and the findings with the tuning forks and the spoken voice were in agreement.

In the entire group of children examined four were found who could hear the whispered voice in both ears at close range. Three were found who could hear the whispered voice in the left ear only.

A rather loud spoken voice was heard as follows:

Ad aurum, bilateral	35 children, or	9%
Ad aurum, right only	42 children, or	10%
Ad aurum, left only	44 children, or	11%

We would like to call attention to the fact that 129 of the children examined have a variable hearing. It often changed after the test was performed and appeared worse if the child was examined in the presence of other children.

Vestibular Tests. Spontaneous nystagmus and fistula tests were negative in all of the children examined. Nystagmus in all three degrees was elicited bilaterally in 288 children, or 70.5 per cent. The reaction in 22, or 5.5 per cent, was a 1 or 2-degree nystagmus and upon repetition presented the same findings.

There was no reaction obtained in 88 cases, or 22 per cent. As was mentioned before, one child out of every four who presented a negative turning reaction also reacted negatively to cold water caloric stimulation. Of the 398 children examined we found that 289 were deaf or had defective hearing on a congenital basis. Of these 289 there were 251, or 87 per cent, who presented positive turning ractions with a 1, 2 or 3 degree nystagmus. Thirty-eight children, or 13 per cent, presented no reaction to turning tests or caloric stimulation.

Of the 63 children, whose deafness or defective hearing was considered as being due to other causes, (i.e., chronic otitis media, etc.) 20, or 33 per cent, presented no reaction to turning. Forty-three, or 67 per cent, presented normal reactions to turning.

In the meningitic group of 46 patients 16, or 32.8 per cent, presented normal reactions and 30, or 67.2 per cent, had negative reactions.

As was mentioned above, 22 children out of the total presented reactions of only first and second degree nystagmus. This amounted to only 55 per cent of the total. These findings were scattered about evenly in the three main groups. The lack of caloric response in 13 per cent of congenitally deaf children is probably caused by the fact that many congenitally deaf patients belong to the group of the hereditary degenerative type. In these cases an equal spreading of degeneration in both labyrinthine nerve distributions was described. It explains the total functional elimination of both parts of the labyrinth.

It is also known that in proven cases of congenital deafness the pars superior of the labyrinth may be affected to some extent. Changes similar to those found in the macula sacculi are often seen in the maculae acusticae and the macula utriculi.

With regard to the meningitic group in which 32.8 per cent presented normal reactions, one must recall the evidence of cases described by Mygind.[5] He found that the pars superior was less affected in a number of cases in meningeal labyrinthitis and that the utriculus persisted without changes.

The postmeningitic group presents some complaints and problems that are not met with in the congenital group. These children were the only ones encountered who complained of "head noises" or "noises in the ears." One or two of them complained bitterly about this difficulty. We could not find a single congenitally deaf patient with this complaint. It is also well known at the school that the children who had meningitis and subsequently became deaf or hard of hearing have difficulty with orientation in space for a variable length of time after the disease. It seems that they have difficulty at night and cannot be trusted to walk in the dark. There have been numerous falls and injuries because of this. Also, they must be watched carefully in a swimming pool until the defect becomes compensated. Falls and injuries are likely to result from a boy running and looking up to catch a ball after having had meningitis and the inner ear complications from it.

Pharynx and Larynx. Tonsils and adenoids were found in 185 children, or 47 per cent, but there was no evidence that adenoids had been responsible for the hearing loss. The eustachian tubes were patent in all cases. Laryngeal examinations were all negative in regard to motor function. Pareses or atrophic conditions of the vocal cords were not noted by mirror examination.

Chronic Otitis. Only six children were found who had had mastoidectomies. Four had had operations on the right ear only, and two had bilateral operations. All of these ears were still discharging and presented active infection with the exception of two. One of these was a simple operation and the other a radical. The remainder of the children with discharging ears had received treatment but it seemed rather ineffective.

Speech. In 82 cases out of the total good usable speech was found to be present. In this group 20.6 per cent were children with large hearing residuals and postmeningeal deafness. Thirty-nine, or 9.8 per cent, had only fair speech. This more liberal term was applied to those deaf children who were able to communicate but did not possess a clear or intelligible speech when tested by the physician. This could hardly be considered usable for general practice. Two hundred and seventy-seven children, or 69.8 per cent,

had very poor or no speech at all. One hundred and fifty of them were in so-called Group II. This was the group made up of hard of hearing children who had enough residual hearing for use in teaching. They, of course, used group hearing aids. They were taught lip reading and their speech was encouraged, corrected, and developed. A good number of these children did not have individual hearing aids. Only 15 possessed hearing aids they could wear at all times.

The remainder of the children were taught by the manual method. They associated with the children who could hear and in this association the means of communication was by use of manual expressions.

There were 22 students who could hear the loud spoken voice at the ear or perhaps one to two feet from the ear but who did not understand words at all. Their speech had not been developed but from the sound of the word spoken to them could repeat it with fair accuracy, especially if it was a very simple word. We do not know whether it would be practical, even with the best hearing aid and individual attention, to attempt instruction of these students by way of the ear. The fact is, more effort must be expended to find out exactly to what extent any of these children hear the spoken voice. One has to be very careful in evaluating the hearing present on the basis of words spoken to them because they just do not recognize words. Here the audiogram in the voice range is of value as a starting point if enough tests are done to obtain an accurate reading. A study in this direction promises to be a grateful field for further development.

SUMMARY AND CONCLUSIONS

In evaluating the present status of examinations and school training of deaf pupils, we find that we are in a period of advancement. Although we agree that the problem of education of the deaf is preferably one of educational agencies and the teaching personnel, we also find that it is an important function of the otologist to help establish an exact diagnosis and to recommend the proper medical procedures. In this regard the progress is indicated by a new policy of the school to admit children only after a complete otological examination and diagnosis related to the type of deafness.

From the otologist's point of view there is still a great necessity for intensive study of the genetics of deafness and an urgent duty to inquire into the marital status and familial relationship more carefully. Family histories are started in the school and forms for

statistics and pedigrees planned. Beginning with this survey efforts have been made, in collaboration with the teaching staff, to establish a precise diagnosis of all types of deafness. The clinical classification of deafness was found helpful in the organization of individual training.

We have learned that the acquired type of deafness causes symptoms not present in the congenitally deaf. Head noises and dizziness are the most annoying of these.

It was interesting to note that in a considerable group (33 per cent) of postmeningeal cases the labyrinthine test revealed normal or subnormal reactions to turning and caloric stimulations.

We observed that a rather small number of students are using a permanent hearing aid. Fitting and testing of instruments cannot be performed locally inasmuch as a hearing clinic is not established in the school or in the vicinity. The pupils have to go to another town in order to be fitted.

In conclusion we would like to stress the fact that it is particularly important in deaf children to reduce the incidence of infections of the upper respiratory tract.

Children with residual hearing should be carefully watched for changes in the nasopharynx after colds. Repeated salpingitis may reduce and damage hearing islands.

Tonsillectomies and adenoidectomies should be performed more often. Children with residual adenoid tissue or recurrent hypertrophy should have another operation. Excess lymphoid tissue on the lateral pharyngeal wall and in the tubal ostium should be treated with radon or x-ray.

Suppurative infections of the ears with extension of the infection toward the mastoid should be treated conservatively for only a relative length of time and then referred to the otological department.

Children with postmeningeal deafness should be advised to choose only occupations in which excessive upward movements of the head can be avoided.

Intensive tinnitus should be classified as to the origin and treated accordingly.

It is our impression that institutions for deaf children with such a large group of various types should be able to arrange an uninter-

rupted otological service. This will help to establish not only a precise diagnosis of the type of deafness, but it will prevent a loss of the residual hearing which is present. It will also secure an improvement in hearing and aspire to a more advantageous use of the latest methods and devices at the school.

We would like to express our appreciation for the cooperation and material assistance of the Superintendent of the Illinois Eye and Ear Infirmary, Mr. Lester Gerber. We are also gratefully obliged to Professor Francis L. Lederer for his suggestions and advice in conducting this survey.

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XLVI

A NEW ANTERIOR COMMISSURE LARYNGOSCOPE

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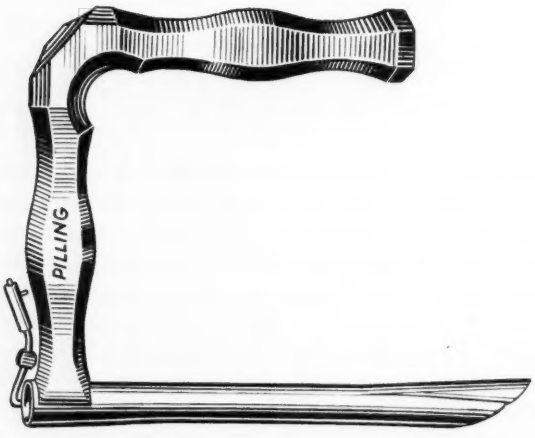
The new anterior commissure laryngoscope herein described uses the principle of construction of the Yankhauer postnasal speculum to facilitate the exposure of the anterior commissure of the larynx. The basic form of the laryngoscope is that of the Jackson anterior commissure laryngoscope modified by incorporating the light carrier in the barrel of the instrument to give a smooth tip, as suggested by C. L. Jackson. The field exposed by this new laryngoscope has been increased anteriorly by elevating the distal two centimeters of the scope diagonally upward and moderately flaring the sides laterally. The proximal opening of the laryngoscope tube has been increased in diameter beyond the actual circumference of the tube, the increase being offset posteriorly. Thus, the maximum advantage of the increased distal diameter of the instrument may be obtained both for the operator's eye and for the direct passage of forceps anterior to the true direction of the lumen of the tube.

The light and the light carrier are placed on the left side of the laryngoscope to facilitate its introduction in patients with protruding teeth. Twin light carriers are available, and while they give an increased and more even illumination, they make the instrument somewhat cumbersome.

The laryngoscope is made in 17 cm. and 18 cm. light carrier lengths for adults and in a 12 cm. length for children.

700 N. MICHIGAN AVENUE.

From the Department of Laryngology, Rhinology and Otology, University of Illinois College of Medicine and St. Luke's Hospital, Chicago.



New laryngoscope with raised distal tip to facilitate exposure of the anterior commissure.

XLVII

DEEP NECK INFECTION

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Any infection in the neck which is, in the beginning, deeper than the skin may be designated deep neck infection. Those infections which start in the skin, such as furuncles, carbuncles, or infected superficial lacerations, are not primarily deep neck infections, as they are at first limited to the skin and its contained structures. By extension, however, these may become deep infections, but run a course of different character. In addition to being regarded as deep in location, deep neck infection is so designated because it originates from a deep source, such as the pharynx, tonsils, salivary glands, temporal bone, vertebrae, nasal sinuses, dental region, deep lymphatic glands, esophagus, and the respiratory passages.

Infections from the various sources mentioned exhibit a variety of manifestations by which they may be recognized. Certain symptoms and signs may be common to some or all of them, for example, painful swallowing and sepsis. Once a severe sepsis is definitely established and persists after reasonable efforts to bring about subsidence with chemotherapy, no time should be lost in applying surgical drainage at its starting point in the neck. If, after such drainage, the sepsis is not controlled, immediate ligation or resection of the jugular vein and its main tributaries should be done, or the outcome will probably be fatal. Thrombosis of the internal jugular vein is the most common result of failure to drain while the infection is still confined to the neck.

For a clear understanding of the subject it is important to know something about the etiology, the applied anatomy, and the regional manifestations.

ETIOLOGY

Etiology is regarded from the two standpoints of bacteriology and portal of entrance.

Bacteriology is of increased importance since the sulfonamide drugs and penicillin have become available for therapy. Briefly, it

may be stated that practically all of these infections are caused by members of the pyogenic group; of these, members of the streptococcus group are most commonly found. The streptococcus hemolyticus appears in pure culture more often than any other organism, occurring in my experience in about 40 per cent of those cases in which cultures were taken and grown. Some one or more of the forms of streptococcus appeared in over 80 per cent of all the infections. Many combinations of bacteria occur. The fusospirochete organisms of Vincent's angina are frequently found in the mouth, particularly when the infection originates in the dental region. In the early stages of such infections, before the neck has been invaded, administration of the antiluetic arsenicals will often bring about a cure[1] and prevent osteomyelitis of the jaw.

The invader of the neck or the blood stream is usually one of the several types found in the mouth or throat and is most often of the streptococcus group, hemolytic or nonhemolytic. In one case in which the upper half of the tonsil had completely disappeared by necrosis caused by the fusospirochete organisms of Vincent's angina the blood stream invader was the streptococcus hemolyticus. It should be remembered that repeatedly negative blood culture reports may be explained by the possibility that the infecting organism may be an anaerobe. In such cases cultures should be grown under both aerobic and anaerobic conditions, and it would not be amiss to make this routine.

The bacteriology has acquired more significant importance since the advent of chemotherapy. The sulfonamide drugs exert their greatest effect on the streptococcus group, particularly the hemolytic type, and to a lesser extent on the staphylococcus group. Their action is of a bacteriostatic or inhibitory nature, the actual bactericidal processes being carried out and immunity established by the cells and fluids of the body, namely, phagocytes and antibodies. It is highly desirable to demonstrate the presence of antibodies in the blood before discontinuing administration.[2] Penicillin acts against gram-positive organisms and is potently active against the staphylococcus group. It is ineffective against gram-negative organisms. Another important weakness of penicillin is that it does not permeate the subarachnoid space in appreciable amounts, and therefore in cases in which the infection has involved the meninges it is necessary to administer the drug intrathecally by lumbar or cisternal puncture and sometimes directly into the ventricles. Penicillin is actually bactericidal and may therefore be used to considerable advantage locally in the wound. The wound may

be flushed frequently through indwelling catheters or drainage tubes. It may be given intravenously as well as intramuscularly. For information as to its exact dosage and indications the reader is referred to an article by Keefer, Herwick, Van Winkle, Jr., and Putnam.[3]

Chemotherapy should not be withheld merely to satisfy curiosity as to the type of the infection. In severe cases time-saving is important. Infection travels rapidly and valuable time may be lost. A safe rule is to drain surgically if there is no improvement after 48 to 72 hours of chemotherapy.[4]

Knowledge of the portal of entrance enables one to anticipate and recognize the pathway of extension into the neck, also sometimes to anticipate and recognize complications, and to determine the place for drainage.

The majority of deep neck infections, about 50 per cent, results from infections in the pharynx and the tonsils. Adenoid infection is included as of pharyngeal origin. When infection enters by this gateway the most frequent place of neck involvement is the pharyngomaxillary fossa. Sometimes infection entering the neck from the region of the lower pole of the tonsil extends downward into the neck along the visceral fascia lateral to the hypopharynx and larynx, and thus causes a visceral, or pretracheal, fascia infection.

When the infection is dental in origin the submaxillary, or Ludwig's angina, type may be anticipated. In my experience about 20 per cent were of dental origin. If infection enters after trauma to the hypopharynx or the esophagus the pretracheal, or visceral, fascia type occurs.

The history is important because in some instances the signs and symptoms at the portal of entrance may have entirely disappeared before the patient is seen with the neck infection. This applies particularly to the region of the tonsil and pharynx. The persistence of a cervical adenitis, particularly in infants, may be a valuable clue as to the side of greater likelihood when there is no visible swelling elsewhere.

The temporal bone with its contained organ of hearing may be the portal of entrance and the manifestations differ according to the part of the temporal bone from which the infection comes. If the infection comes from the cells in or near the mastoid tip, there results a condition called Bezold's abscess. It is an abscess deeply situated, anterior to the mastoid tip, between it and the mandible, and extending inward toward the pharynx and downward into the neck

under the sternomastoid muscle, and forward and inward along the digastric muscle to the submaxillary triangle. The parotid gland is also involved to a greater or lesser degree. The external auditory canal is also more or less involved and may actually become one of the points of drainage by spontaneous rupture. The usual point of exit of the pus from the mastoid cells is through the cortex of the medial aspect of the mastoid tip and through the digastric groove.

An increasing number of reports of infection of the pharyngomaxillary, or parapharyngeal, region secondary to infection of the petrous part of the temporal bone is appearing in the literature. This extension takes place through the inferior aspect of the petrous pyramid, actually a part of the skull base. Draining the pharyngomaxillary space in such cases often also drains the petrous pyramid abscess. Symptoms and signs in such a case are a combination of petrous pyramid suppuration and pharyngomaxillary neck infection.

There is also evidence that the petrous pyramid apex may be infected by the spread of infection from the sphenoid sinus, perhaps also from the ethmoids, and that a pharyngomaxillary fossa infection may result when the infection extends through the inferior aspect of the bone into the pharyngomaxillary space. Mosher[5] has reported a case with such a roundabout pathway of travel. In the case reported there was also infection of the mastoid cellular structure requiring mastoidectomy. Probably many such cases escape detection. Drainage of the pharyngomaxillary fossa brought about cure of the petrous pyramid infection.

Infection in the zygomatic cells of the temporal bone is the source which involves the bone structures entering into the formation of the roots of the zygoma and the articular surface of the temporomandibular joint, the glenoid fossa. From this source abscess forms in the zygomatic and pharyngomaxillary fossae and sometimes also in the pterygomaxillary and temporal fossae. In these the parotid gland may also be involved.

Facial nerve paralysis is one of the rarest of complications in these acute suppurative conditions involving the mastoid cells and the soft parts contiguous to the stylomastoid foramen. In fact I have never observed it in the presence of Bezold's abscess. The best explanation that occurs to me is that because of the tissue softening that results from abscess formation, the nerve is not subjected to any pressure. It is known that a nerve graft accurately placed in the fallopian canal may heal despite the fact that it may be bathed with pus.

Infection of the nose, nasal sinuses and nasopharynx sometimes causes abscess of the uppermost retropharyngeal lymph nodes situated at the level of the second cervical vertebra. This constitutes the well-known retropharyngeal abscess which can in most cases be drained by peroral incision. It usually occurs in infants and young children.

Infection from caries and necrosis of the bodies of the cervical vertebrae is at first an infection beneath the prevertebral fascia and does not involve the retropharyngeal space until it penetrates the prevertebral fascia. It is most often lateral, and not medial, in location and lies posterior to the great vessels in the pharyngomaxillary space. It is tuberculous in the majority of instances.

Infections originating in the salivary glands are not uncommon[6] and in the majority of instances are accompanied by calculus formation which may be primary or secondary. The same type of inflammation may not be accompanied by calculus formation. The salivary gland inflammation may itself be secondary to dental disease. The symptoms vary according to the gland involved and manifest themselves by inflammatory swelling, discomfort and pain in that gland. Secondary systemic parotitis may occur during severe abdominal or pelvic disease or after operation on abdominal or pelvic viscera.

Infections of the hypopharynx, esophagus, larynx, and trachea may extend into the neck and cause visceral, or pretracheal, fascia infection. Trauma to the hypopharynx and esophagus by unmasticated food, foreign bodies and endoscopic instruments is a common cause. Visceral fascia infection may also be caused by infection in and about the tonsils. Visceral, or pretracheal, fascia infections are the most dangerous of all types of deep neck infection, frequently causing mediastinal suppuration by extension downward into the chest. Internal or external injuries involving the larynx or the trachea also cause neck infection in which there may occur emphysema. This emphysema may be from air which escapes into the tissues of the neck, or it may be an infectious emphysema caused by gas-producing bacteria. Fractures of the laryngeal and tracheal cartilages may be followed by infection and emphysema.

Infection of the deep cervical lymphatics may accompany all types of infection of the neck, but may exist without any other evidence of infection in the neck. Infection present in the tonsils or the teeth may pass into the glands of the neck through the lymph channels without involving the intervening structures between these two

areas. In such cases, for a time, the cervical adenitis constitutes the whole of the neck infection.

Congenital cysts and fistulae may also be the cause of deep neck infection when suppurative inflammation occurs in them.

Mediastinal suppuration and empyema high in the chest may involve the neck secondarily.

A goodly number of infections follow surgery of the tonsils and adenoids, as may be noted by the numerous reports in the literature over the years. These occur more often after local than after general anesthesia.

Dental disease is frequently the forerunner of severe deep neck infection. Infection may result from disease of the crown or the root of a tooth, or from pyorrhea alveolaris. It may also result from gingivitis alone. Neck infection from the dental region most frequently follows dental extractions, particularly after the use of local anesthetics by needle injection. The involvement may occur along the tract of the needle. The dental infection may therefore be said to be unprovoked or provoked. The former refers to those in which no surgical dentistry has been applied, and the latter to those that follow extraction, cavity filling, or needle injection. Filling a cavity in a tooth which has an apical suppuration is almost certain to be followed by a severe infection which may rapidly extend into the neck. Most of the neck infections of dental origin result from a source in the teeth of the mandible, though infection may also originate in the teeth of the maxilla. Clinically the pictures are entirely different.

In infection from the mandibular region the inflammation involves the submaxillary, submental and parotid regions, and the upper part of the cervical region adjacent to these triangles. There usually results the typical picture of Ludwig's angina. There may also occur osteomyelitis of the mandible. Symptoms and signs differ according to whether the dental infection extends along the lingual or the labial side.

If the infection advances along the lingual side, it first involves the floor of the mouth and the tongue base, thus reaching the vicinity of the internal pterygoid muscle on the inner side of the body, angle and ramus of the jaw, all of which causes a pronounced trismus with swelling of the tongue, crowding that organ backward toward the pharynx and upward into contact with the soft palate. Mastication, swallowing, and even breathing are thus embarrassed.

At such a time the infection has already gone beyond the mylohyoid muscles and involved the neck in the submental, submaxillary, and adjacent upper cervical regions and constitutes the well-known Ludwig's angina.

If the infection travels along the labial side of the mandible, the floor of the mouth and the tongue base will not be involved and there will be little or no trismus and no embarrassment of swallowing or respiration. There may be a slight trismus with painful mastication from inflammation in and about the masseter muscle. If there is extension into the neck, the infection passes over the outer surface and lower border of the jaw by continuity.

Infection not infrequently takes place along the pathway of the needle when mandibular nerve block is done. In such instances the infection of the neck involves the pharyngomaxillary region and the symptoms and signs are those characterizing infection in that region.

Infections from the teeth of the maxilla may likewise travel along the lingual or the labial side. If the pathway is on the lingual side, the roof of the mouth will be involved, with little or no likelihood of neck infection.

If the pathway is on the labial side, dire consequences may result. The entire side of the face may be quickly involved, with extension downward over the upper cervical region on that side. At the same time the infection may travel backward behind the maxilla to the sphenomaxillary, zygomatic and temporal fossae, and upward into the region of the apex of the orbit and sphenomaxillary fissure. When the infection has reached such a stage, the pharyngomaxillary fossa has also become involved. The maxillary sinus eventually becomes involved. When the infecting tooth is in relation with its floor, infection of the sinus is probably present before the infection has extended to these other parts.

APPLIED ANATOMY

In his Presidential Address to the American Academy of Ophthalmology and Otolaryngology in the year 1929, Dr. Harris P. Mosher[7] announced the submaxillary fossa approach to deep pus in the neck, and in the paper incorporated much on the applied anatomy of the neck. It is, I believe, the most important emergency procedure in neck surgery since the perfection of the tracheotomy.

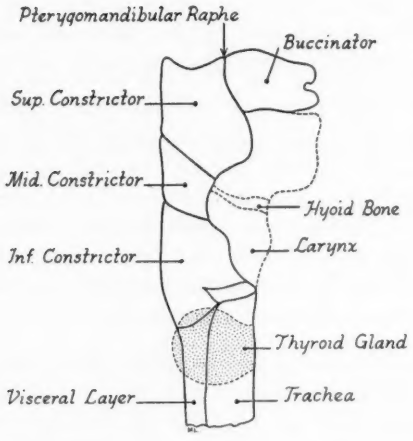

Fig. 1.—Diagrammatic drawing of visceral fascia (buccopharyngeal and pretracheal).

His description of the applied anatomy of the cervical fascia is the essence of simplicity. On that foundation much of what is here set down rests. He stated, in effect, that one may assume that all the principal layers of the deep cervical fascia stem from, or take origin from, the carotid sheath, and that infection anywhere in the neck may therefore reach the sheath, in which event thrombosis of the internal jugular vein is always a possibility. The logical reasoning from this is that the carotid sheath may be exposed and used as an avenue of search for pus deep in the neck.

Death from neck infection is the result of one or more of three main causes, namely, septicemia, asphyxia, and hemorrhage. Septicemia is the most common cause of death. Secondary infection in one or more of the vital organs may be the direct cause of death.

The neck is that part of the body which comprises all of the anatomical structures intervening between the base of the skull and the upper end of the chest cage. It may be divided into two portions: one, quite the more massive, is made up of the cervical spine and its attached musculature; the other portion, smaller and situated anterior to the larger mass, may be termed the visceral portion. It is in this visceral portion that almost all deep neck infections occur. The exception is the rarer instance when the infection starts in the

cervical vertebrae and spreads into this visceral portion. The osseomuscular portion has the functions of support for the head and the housing of the cervical portion of the spinal cord. The visceral portion accommodates all of the neck viscera, vessels, nerves, lymphatics, all of which are held in their proper relationships by the various layers of the cervical fascia.

In order to clarify the terminology used in this text a short description of the anatomy of the triangles of the neck is incorporated. The side of the visceral part of the neck may be regarded as a quadrilateral area which is continuous at the midline of the neck with a similar quadrilateral area on the other side; in other words, two quadrilateral regions which are in juxtaposition along one of their sides.

Each quadrilateral area is divided into two triangles by a diagonal represented by the sternomastoid muscle. One of the triangles thus formed has its base above along the lower border of the jaw and its apex down at the upper end of the sternum, and is known as the anterior cervical triangle. The other, the posterior cervical triangle, has its base at the upper border of the clavicle between the trapezius muscle behind and the sternomastoid in front. Its apex is above at the occipital bone between the same muscles.

Each of these triangles is again divided into subordinate triangles by certain muscles. The posterior triangle is divided into two triangles, a superior and an inferior, by the inferior belly of the omohyoid muscle. The upper triangle is called the occipital triangle and the lower one the subclavian triangle. The anterior cervical triangle is subdivided into four triangles by the superior belly of the omohyoid muscle and the two bellies of the digastric muscle. The two bellies of the digastric muscle form a triangle which has its base along the lower border of the jaw and its apex at the central or intermediate tendon which is attached to the hyoid bone. This is called the submaxillary triangle.

The superior tendon of the omohyoid muscle divides the remaining space into two triangles, one above it and one below it. The upper of these triangles has its base posteriorly along the sternomastoid muscle. Its other two boundaries are the posterior belly of the digastric above, and the superior belly of the omohyoid below. Its apex is at the hyoid bone at the junction of these two muscle bellies. It is called the superior carotid triangle.

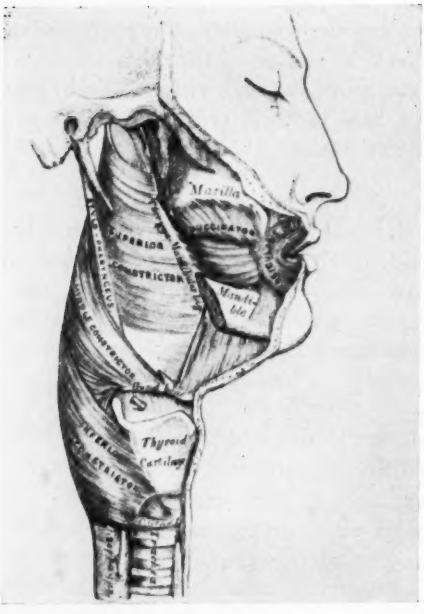

Fig. 2.—Muscles covered by the buccopharyngeal fascia. Also shows the pterygomandibular raphe. (From Gray's Anatomy, used with permission of the publishers, Lea and Febiger.)

The third triangle lies below the superior belly of the omohyoid. Its base is along the median line from the hyoid bone to the sternum. Its other two borders are the superior belly of the omohyoid above and the anterior border of the sternomastoid below. It is called the inferior carotid triangle. Its apex lies posteriorly at the anterior border of the sternomastoid.

Another triangular area not included in the aforementioned lies between the jaw symphysis and the hyoid bone. Its base is below at the hyoid bone and its apex is at the chin in the midline. Its sides are formed by the anterior bellies of the two digastric muscles. It is called the submental triangle. It is sometimes considered to be divided by the midline of the neck into two triangles. The simpler conception is to consider it as one triangle, as described.

A working knowledge of the applied anatomy of the neck resolves itself into a consideration of (1) the layers of the deep cervical

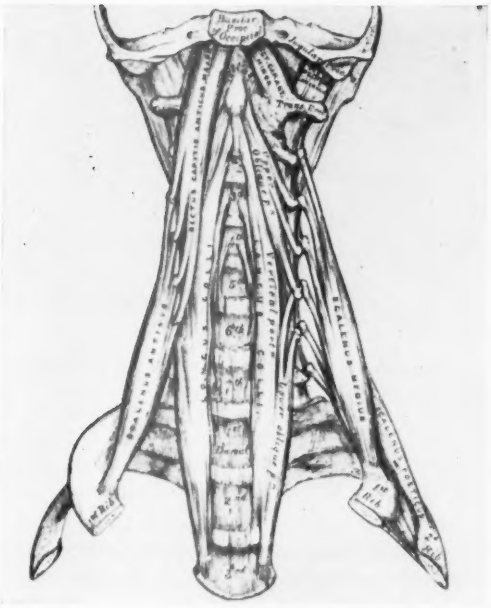

Fig. 3.—Structures covered by the prevertebral fascia. (From Gray's Anatomy, used with permission of the publishers, Lea and Febiger.)

fascia, (2) the compartments formed by the arrangement and the attachments of these layers, (3) the arterial and venous vessels, and (4) the anatomical arrangement of the cervical lymphatics. Knowledge of the applied anatomy is important because a better understanding of the behavior of infections is thus made possible and also a better understanding of where drainage should be placed. This is particularly true if the portal of entrance of the infection is known.

The cervical fascia is customarily described as being composed of superficial and deep layers. The superficial cervical fascia is a simple enveloping layer, intimately connected with the skin, and therefore enveloping the entire neck. It is of minor importance in the problem of deep neck infection. Over the triangles of the neck it may be said to contain, within itself, the platysma muscle. The superficial cervical lymph nodes are in relation with this fascia.

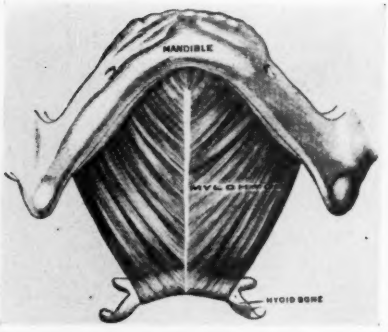

Fig. 4.—This muscular diaphragm separates the mouth from the neck. The submaxillary salivary gland bends upward and inward around its free posterolateral border. The gland is therefore situated partly in the neck and partly in the mouth. (Poirier and Charpy)

The deep cervical fascia is composed of several layers, the most important of which are mentioned in this description. These layers are (1) the superficial layer of the deep fascia, or splitting layer; (2) the prevertebral layer; (3) the visceral fascia, which is composed of the buccopharyngeal and the pretracheal fasciae; (4) the carotid sheath fascia; (5) the parotid fascia; (6) the submaxillary gland fascia; and (7) the thyroid gland fascia. These are the layers of the deep cervical fascia which are significant in deep neck infection. The temporal fascia of the head occasionally requires consideration when infection from the maxilla or from the zygomatic cells of the temporal bone involves the zygomatic and temporal fossae.

The superficial layer of the deep fascia completely envelops the neck, reaching from the skull to the chest. It splits to envelop the trapezius and sternomastoid muscles. In the front of the neck it takes firm attachment to the hyoid bone. From the hyoid bone it continues upward, splitting to envelop the submaxillary and parotid salivary glands. The superficial layer of this splitting takes attachment on the lower border of the mandible, covers the masseter muscle and the parotid gland, and becomes attached to the zygoma. The deep layer of this splitting, beneath the salivary glands, is attached to the mylohyoid ridge on the inner surface of the mandible, and, laterally, to the styloid process and the inferior surface of the mastoid process. Beneath the parotid gland and between the parotid

and submaxillary glands the union of these layers which connects the styloid process with the angle of the jaw is thickened to form a ligament called the stylomandibular ligament. The deep part of the parotid gland, which extends inward toward the pharynx and is called the retromandibular or pharyngeal process of the parotid gland, is devoid of fascial covering at its innermost part where it is in relation with the pharyngomaxillary space. The posterior or lateral end of the submaxillary salivary gland bends upward and inward around the free posterolateral border of the mylohyoid muscle, and comes into relationship with the floor of the mouth. Thus is established a direct relationship between the mouth and the neck, the mylohyoid muscles being the normal boundary between the two regions.

Beneath the sternomastoid muscle the superficial layer of the deep fascia is in direct contact and relationship with the superficial aspect of the carotid sheath. The deep aspect of the sheath is in relationship with the prevertebral fascia. Where this deep relationship exists over the anterior surface of the scaleni muscles and the brachial plexus, it is of considerable significance because infections may travel along these muscles and nerves to the costocoracoid and axillary regions. The prevertebral fascia encircles and covers the spinal column and the muscles attached thereto, reaches from the skull base to the chest, and is continuous into the posterior mediastinum. Anterior to the body of the second cervical vertebra on each side of the median line there are one or more lymph nodes which take lymph drainage from the nasopharynx, nose, and nasal sinuses. Abscess frequently forms in these glands causing retropharyngeal abscess.

The visceral fascia envelops the viscera of the neck, namely, the pharynx, esophagus, larynx, trachea, and thyroid gland. Where it covers the constrictor muscles of the pharynx and the buccinator, it is known as the buccopharyngeal fascia. The buccopharyngeal fascia reaches from the skull base to the cricoid cartilage where the inferior constrictor takes partial attachment. Along the line of this fascia, where both the superior constrictor and the buccinator are attached, is a thickening called the pterygomandibular raphe, and this marks the anterior limit or boundary of the pharyngomaxillary fossa. It is in the vicinity of this ligament or raphe that infection may enter the pharyngomaxillary fossa after attempted blocking of the mandibular nerve. The superior constrictor muscle, which is thin, forms a large part of the wall of the tonsil fossa and separates the tonsil fossa from the pharyngomaxillary space. Therefore, infection from the tonsil, which penetrates the superior constrictor muscle, invades

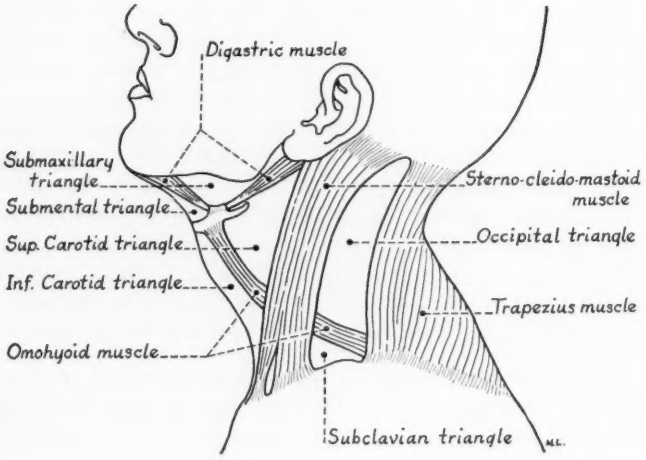

Fig. 5.—Drawing showing the triangles of the neck.

the pharyngomaxillary fossa. From 60 to 70 per cent of pharyngomaxillary infections are of tonsillar or pharyngeal origin. The superior constrictor muscle forms the median wall or inner boundary of the pharyngomaxillary fossa.

Where the visceral fascia covers and envelops the trachea and esophagus it is known as the pretracheal fascia. In the fetus, as the primitive thyroid gland descends into the neck and becomes located beneath the infrahyoid muscles in front of the trachea, it has become surrounded by a layer of fascia, the thyroid gland fascia. This fascia is intimately connected with the pretracheal fascia, in fact so much so that for practical purposes it may be considered a part of it.

Between the central, or intermediate, tendon of the omohyoid muscle and the upper end of the sternum the layer of cervical fascia which envelops the infrahyoid muscles is thickened into a band which binds the central tendon of the omohyoid muscle to a fixed point on the clavicle and the first rib. Sometimes when it becomes desirable to expose the carotid sheath throughout its entire length in the neck it may be necessary to divide the superior belly of the omohyoid muscle.

The carotid sheath fascia, as previously stated, is in relation with the splitting layer on its superficial aspect and with the prevertebral

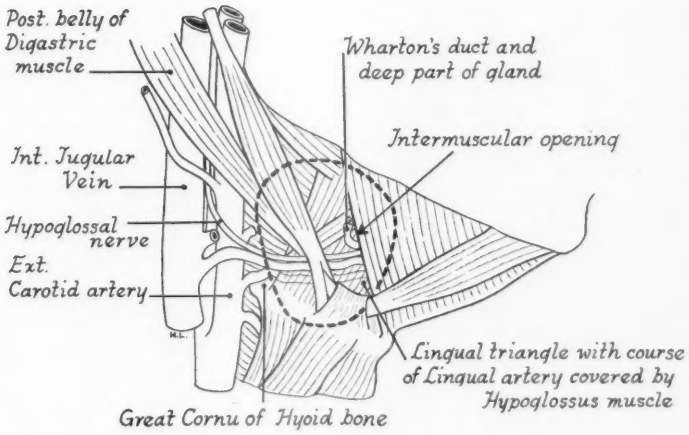

Fig. 6.—Drawing showing the intermuscular opening at the posterior border of the mylohyoid muscle, which is occupied by the submaxillary salivary gland. It may become an avenue of travel for pus from the mouth to the neck. The dotted circle outlines the position of the submaxillary salivary gland. (After Testut)

fascia on its deep aspect. From the skull base it extends downward through the pharyngomaxillary fossa into the neck and chest. It is not only in contact with infection involving the pharyngomaxillary fossa high up in the neck, but may serve as the pathway of extension to the lower part of the neck. In such infections some part of the internal jugular vein is at all times in contact with the infectious process.

The parotid and submaxillary gland fasciae, as previously mentioned, are formed by a splitting of the superficial layer of the deep fascia above the hyoid bone. The parotid gland, on its deep aspect, is in relation with the pharyngomaxillary fossa. The submaxillary gland is, for the most part, situated in the neck, but its lateral or posterior end bends around the free part of the posterolateral border of the mylohyoid muscle and comes into relationship with the floor of the mouth above the muscle. The posterior end of the gland thus lies in an intermuscular opening through which there is a pathway of travel for pus from the mouth to the neck.

The thyroid fascia envelops the thyroid gland and is of minor importance in the problem of neck infection. Infection may be secondary to lymphadenitis.

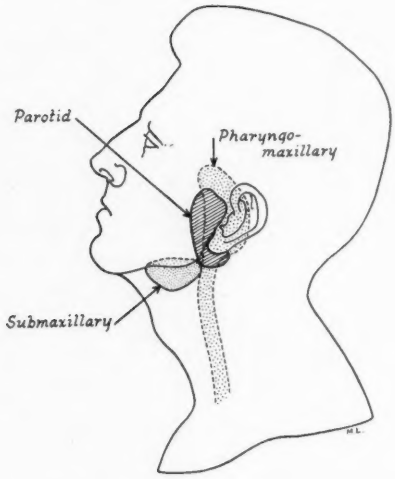

Fig. 7.—Drawing showing the location of fascial compartments in the upper part of the neck, namely, submaxillary, parotid and pharyngomaxillary.

High in the neck, and formed by these fascial layers, there are three important fossae or compartments. These are (1) the pharyngomaxillary, (2) the parotid, and (3) the submaxillary. The parotid and submaxillary compartments contain the respective salivary glands and some important lymph nodes. The pharyngomaxillary fossa is not an actual space except when distended with pus or blood. Under normal conditions it may be said to be a potential space. It has, however, very definite boundaries. It is somewhat cone-shaped with its base uppermost at the base of the skull. Its apex is below at the hyoid bone. It is situated lateral to the pharynx, and its medial, or inner, boundary is the superior constrictor muscle. Its superior boundary is the base of the skull. Its lateral, or outer, boundary is formed by the parotid gland, the mandible and the internal pterygoid muscle. Its posterior boundary is the prevertebral fascia. Its anterior limit is the pterygomandibular raphe. It is divided into two unequal parts, anterior and posterior, by the structures which are attached to the styloid process. The posterior part contains the vessel sheath and the cranial nerves which emerge from the foramina in the skull base. On its medial side, the anterior compartment is in close relationship with the tonsil fossa; on its lateral

side, with the internal pterygoid muscle. Infection in this anterior compartment is characterized by displacement inward of the tonsil and fauces and by marked trismus. Infection in the posterior compartment may cause little or no trismus, but there is swelling of the lateral pharyngeal wall and perhaps of the posterior pillar of the fauces. With infection in either of the compartments there may be swelling of the parotid gland, noticeable externally.

Infection of the carotid sheath and its contents, with or without thrombosis of the internal jugular vein, is a frequent complication of infection in the pharyngomaxillary fossa. A severe sepsis is strong indication that thrombosis is present or imminent. Erosion of the internal carotid artery sometimes occurs; this results in fatal hemorrhage when it breaks through into the pharynx. Formation of so-called pseudo-aneurysm by laminated blood clotting takes place prior to this rupture. Peroral incision through the lateral wall of the pharynx is definitely contraindicated in such cases, as the resulting hemorrhage could not be controlled. Swellings which are the result of extravasated blood should not be mistaken for abscess. The surgical approach must be made externally through the neck where vessel ligation can be performed at the same time. Repeated hemorrhage of small amounts of blood into the pharynx should arouse suspicion that a vessel is eroded. Erosion of the walls of the vein is usually accompanied by thrombus formation and not by hemorrhage. The walls of the vein may have completely disappeared for a short distance and the course of the vessel outlined by a thrombus. Occasionally the sheath is filled with pus, having the appearance of a more or less distended sac.

Batson[8] has aptly stated that the neck is composed of tissue masses (bones, muscles, viscera, blood vessels, etc.) held together by connective tissues or fasciae; that these fasciae are capsular, areolar, and very densely perivascular; and that the perivascular fasciae with the vessels form pedicle-like structures connected with the neck viscera. The carotid sheath with its contents forms the largest of these pedicle-like structures.

The lymphatics of the head and neck are divisible into two main groups, a superficial and a deep.

The superficial glands of the neck are in relation with the superficial fascia and are sometimes numerous and sometimes scarce. The most numerous are found along the external jugular vein. There are a few in the upper part of the occipital triangle, some along the anterior jugular veins, and some in front of the trachea. They drain

into the deep glands along the internal jugular vein. There is no very definite arrangement.

The deep cervical glands are situated along the carotid sheath from the skull base to the chest. They are the most important group of lymphatics in the neck and receive the drainage from all the lymphatics of the head and neck. They are divisible into two groups, the superior deep cervical glands and the inferior deep cervical glands. The superior, or upper, group extends to the level where the omohyoid muscle crosses the internal jugular vein. The inferior, or lower, group lies below this level. The glands which are in relation with the brachial plexus of nerves belong to this chain. Those glands just above the clavicle are also called the supraclavicular group. These deep glands on the left side of the neck drain into the thoracic duct; those on the right side, into the right lymphatic duct. They are also in series with the thoracic, or mediastinal, glands. There are several glands in the suprasternal space of Burns.

The lymphatics of the head and the upper part of the neck are arranged in groups along a more or less horizontal line reaching from the occipital region to the chin. These groups are the suboccipital, the postauricular or mastoid, the preauricular and parotid, the submaxillary, and the submental. Their efferent vessels connect with the glands on the carotid sheath, making, with the deep sheath glands, a pattern similar to that made by the ribs and handle of a fan.

Small groups are also located over the face. One group, deeply situated and lateral to the pharynx, is called the internal maxillary group from its position in relation with the internal maxillary artery. The submaxillary group is an important group and receives drainage from the cheeks, lips, mouth, tongue and teeth. Other lymph channels from these same regions connect directly with the deep carotid sheath nodes, by-passing the submaxillary nodes. Others from the mouth connect with the submental nodes, efferents from which pass to the submaxillary and upper deep cervical nodes. Some channels from the tip of the tongue connect directly with glands in the inferior deep cervical group. Most of the submaxillary nodes are in relation with the capsule. The parotid group is next in importance and the nodes are situated under the parotid fascia, many being imbedded in the gland structure. The preauricular nodes are superficial to the parotid gland fascia. Several rather important lymph glands are found in the retropharyngeal space on either side of the median line at about the level of the second cervical vertebra. These are called the retropharyngeal glands, and when

they are infected cause retropharyngeal abscess. This occurs most frequently in infants and young children.

There are deep glands scattered along the lateral walls of the pharynx called lateral pharyngeal glands. They receive drainage from the pharynx and posterior part of the mouth, palate, and tongue. The posterior part of the nasal chambers and the posterior nasal sinuses drain into the retropharyngeal glands. There are glands in relation with the larynx, trachea, and thyroid gland, drainage from which goes to the glands on the sheath. When the glands on the carotid sheath become inflamed they become quite adherent to the sheath and it is probable that blood stream infection may result. There is usually a small gland on or near the cricothyroid membrane.

Above the hyoid bone the lymphatic channels from the submental and submaxillary glands turn sharply in a lateral direction to connect with the glands of the deep chain along the carotid sheath.

When it is recalled from which structures symptoms and signs are derived, from the viewpoint of applied anatomy, the diagnosis is often considerably clarified. The structures giving rise to such symptoms and signs are the lymphatic vessels and nodes, the blood vessels, the nerves, the cervical viscera, and the muscles. From involvement of the lymphatics may be expected high continuous temperature, and this sometimes without much swelling visible on the surface. From involvement of the veins may be expected high elevations of temperature with abrupt drops, with or without chills. Frequently there are periods of sweat drenching. Nerve involvement causes pain in the case of sensory nerves, and paralysis in the case of motor nerves. Pupils, pharynx, palate, tongue, larynx, and shoulders may be paralyzed. Dysphagia, odynophagia, dysphonia, aphonia, and dyspnea are indications of visceral involvement. Spasm or splinting of muscles and myalgia occur when the inflammation reaches their vicinity. The internal pterygoid and sternomastoid muscles, producing trismus and torticollis respectively, are notable examples. There may be an actual inflammatory process of the muscles.

Recognition of symptoms and signs resulting from involvement of these structures is helpful in locating the site of the infection, and it is also helpful to know the exact portal of entrance. One may then anticipate the probable pathway of the infection even when, as often happens, there takes place a lymphatic jump to distantly situated glands. To such symptoms and signs as are already present there may be thus added a lymph gland abscess. In other instances

the glands may not produce an abscess but may be the seat of severe acute inflammatory swelling, and if they are in relation with the carotid sheath, may cause an internal jugular vein infection, with or without thrombosis. This markedly increases the gravity of the disease by adding a blood stream infection. The lymph gland infection may result directly from the inflammation of the upper respiratory tract mucosa or tonsils, and thus constitute the whole phenomenon in the neck; or the gland infection may not occur until after the infection has already involved one or more of the neck regions by continuity. It is important not to assume that the cervical adenopathy is the sole pathologic process. In most instances it is only a part of the process.

GENERAL OR SYSTEMIC SYMPTOMATOLOGY

While there is considerable similarity in the symptoms and signs of the several varieties of neck infection, there is also a great variability in the character and the intensity of the manifestations.

The condition known as sepsis is present to a greater or lesser degree in all types of neck infection and it is therefore necessary to define it. The following description applies; apathy, pallor, moist or sweaty skin surface, weakness, poor appetite and indigestion, fever, especially with abrupt rises and drops, nightime or daytime sweating, septic blood count with an increased percentage of young forms, rapid thready pulse of low volume but perhaps full and bounding when the temperature is high, albumin and perhaps casts in the urine, and chills. A positive blood culture and metastatic abscesses are conclusive evidence of blood stream infection. Metastatic infections of distant parts constitute complications and add symptoms and signs referable to those parts.

Frequent blood counts are important. When the sepsis is severe it is advisable to do a complete blood count every 24 to 48 hours. Considered with the local and general condition of the patient it gives valuable information. The total white cell count is almost always elevated, ranging from normal to 40,000. The percentage of polymorphonuclears has its normal variation, depending on the age of the patient, being lower in children. Polymorphonuclear counts may run as high as 95 per cent. A high total white cell count with a moderately increased percentage of polymorphonuclears is regarded as a more desirable condition than the reverse. In the early stage of the infection when the sepsis is severe, the eosinophiles disappear from the blood count. As the patient improves with lessen-

ing of the sepsis, they return. A reappearance of eosinophiles is a sign of improvement. It is said that increase in the percentage of large mononuclears is indicative of walling-off of the infected area.

The percentage of the young forms of the polymorphonuclears, when counted as one group, is usually increased in the presence of sepsis. When this nonsegmented group amounts to more than 15 per cent, it indicates that the toxemia is becoming severe enough to cause noticeable irritation or disturbance of the bone marrow. In the very severe degrees of sepsis the percentage runs as high as 40 or more. In one of my patients there was a young form percentage of 63, with a total white count of 36,050, and a polymorphonuclear percentage of 90.5. When the young forms are as high as 40 to 50 per cent or more, one may expect to see a few metamyelocytes or myelocytes, the appearance of which is always of grave significance. Sometimes the total white count will drop from a previously high mark to normal or slightly less, with no improvement of the patient clinically. In such instances the polymorphonuclear percentage and the young form percentage can be very illuminating. In one of my patients the total white count was 7,200, polymorphonuclears 50 per cent, young forms 35 per cent, eosinophiles 0 per cent, with a fever, at the same time, of 105.4° F. No myelocytes were reported. The child died. In another patient a count was made six days before admission to the hospital and was as follows: total white count 23,900, polymorphonuclears 64 per cent, young forms 30 per cent. On the day of admission to the hospital the total white count was 6,500, polymorphonuclears 35 per cent, young forms 57 per cent, with one basophilic myelocyte. After 48 hours the total white count was 14,800, polymorphonuclears 44 per cent, young forms 53 per cent. This child received extensive drainage and recovered.

In all severe neck infections which I have seen there has been a well-marked secondary anemia. I have not seen a marked leukopenia in a neck infection. Considering that agranulocytosis is frequently accompanied by mouth and throat inflammation, it is possible that a deep neck infection might occur. It is important to recognize a low blood volume concentration, resulting from dehydration. A hemoglobin percentage over 100 with a high red cell count should arouse such a suspicion and may be an indication of blood concentration.

PHARYNGOMAXILLARY FOSSA INFECTION

About 50 per cent of neck infections occur in the pharyngomaxillary fossa, and the most frequent cause is infection from the tonsils and pharynx. The adenoid region is included as pharyngeal.

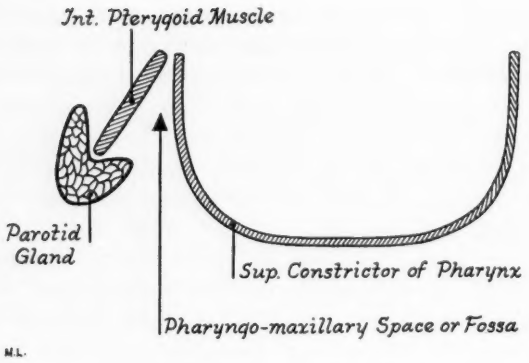

Fig. 8.—Drawing explaining the triad of signs resulting from infection in the anterior compartment of the pharyngomaxillary fossa.

Therapy with sulfonamides and penicillin has reduced the incidence of this particular type of infection. When tonsillitis is treated early with chemotherapy, complicating infection in the pharyngomaxillary space is less likely to occur. When deep neck infection occurs after extraction of teeth the invasion takes place rapidly, resistance is quickly overcome, and chemotherapy is not apt to be administered early. Therefore infections originating in the dental region may be expected to show a relative increase and statistics of the future will change. If before dental extraction is done the patient receives preliminary chemotherapy, infection from this dental source will be decreased. As soon as infection has penetrated the tonsil fossa muscle bed it has become pharyngomaxillary. This applies particularly to peritonsillar abscess which frequently spreads by this pathway. A large solitary abscess may form in the pharyngomaxillary fossa, or there may be one or more small abscesses. Sometimes these smaller abscesses extend downward along the buccopharyngeal fascia and become a buccopharyngeal and pretracheal fascia infection. In the latter condition the tissues along the pretracheal fascia become greatly thickened, and there may or may not be abscess formation along it. Occasionally, during a tonsillectomy, after the tonsil has been separated from its bed, there is seen a fairly large recess lined with granulation tissue, somewhat resembling a cyst, the walls of which are formed by pyogenic membrane. Such a cavity is the result of a former acute peritonsillar infection—and perhaps pharyngomaxillary fossa infection which localized and became quiescent.

There are several avenues of extension from the tonsillar and pharyngeal regions into the pharyngomaxillary space and the internal jugular vein. There can be no doubt that direct extension of the inflammation by continuity of tissues occurs. Thrombosis of the peritonsillar veins, extension by way of the lymphatics, and perivascular extension from single or multiple abscesses in the peritonsillar region are all possible pathways. Because of this tendency to extend in or along vascular channels, thrombosis of the internal jugular vein and blood stream infection are always strong possibilities.

It has been repeatedly observed that the pharyngitis or tonsillitis or both may have, to all appearances, completely subsided before the pharyngomaxillary infection manifests itself. This interval may be as long as several weeks. The history of an antecedent sore throat or upper respiratory infection is important and should not be ignored.

The symptoms and signs indicating infection in the pharyngomaxillary fossa differ according as to whether the infection is located in the anterior or in the posterior part of the fossa. The first manifestations, regardless of location, are fever and painful swallowing or sore throat. Sepsis of greater or lesser severity is present from the start.

If the infection is confined to the posterior part of the fossa, the retrostyloid part, there will be swelling or prominence of the lateral pharyngeal wall, with, perhaps, swelling of the posterior pillar. With this there may also be some swelling of the parotid gland region externally at the level of the angle of the jaw. There will, however, be little or no trismus, and no protrusion or prolapse of the tonsil. As soon as the infection involves the anterior compartment there will be trismus and prolapse of the tonsil and fauces, and in such an instance the infection will be in relationship with the tonsil fossa medially and with the internal pterygoid muscle laterally. There may also be swelling of the parotid gland by involvement of its retromandibular, or pharyngeal, process which is in relation with the fossa laterally. This triad of signs, (1) prolapse of the tonsil and fauces and swelling of the lateral wall of the pharynx, (2) trismus, and (3) parotid region swelling, constitutes conclusive evidence of a pathological process in the pharyngomaxillary fossa. When peritonsillar abscess is present primarily, it may be difficult to determine exactly when the infection has involved the pharyngomaxillary fossa. The appearance of swelling in the parotid region would be strongly suggestive, as this is not as prominent with quinsy alone. Inflamed

and swollen lymph nodes near the angle of the jaw may simulate parotid swelling and should be differentiated. With ordinary uncomplicated peritonsillar abscess the fever does not usually run high and sepsis is not severe. The striking feature is the extreme misery of the patient and the local discomfort in the throat. With acute peritonsillar inflammation there is acute inflammatory swelling of the tonsil itself and edema of the soft palate and uvula. There is often, also, exudation and sloughing in the crypts of the tonsil and sometimes large necrotic ulcers involving a considerable part of the tonsillar surface and substance. In pharyngomaxillary infection not secondary to peritonsillar abscess the tonsil itself may be neither swollen nor inflamed, and there may be no redness of the fauces or pharynx. In both conditions there is trismus. Patients with quinsy seem to me to have much greater difficulty in clearing the mouth and throat of accumulated saliva and secretions, largely because the secretions are more copious and the musculature more acutely inflamed. Those cases in which the peritonsillar abscess is located behind or near the lower pole of the tonsil more nearly resemble pharyngomaxillary infection.

Every peritonsillar infection is a potential pharyngomaxillary infection. If, during the course of a peritonsillar abscess, the fever begins to spike, with, perhaps, sweats and chills, it will be reasonably good evidence of pharyngomaxillary infection and perhaps blood stream infection. In such an event unilateral tonsillectomy, with or without search for the pharyngomaxillary abscess, is a rational procedure. This uncaps the peritonsillar abscess, and gives a view of the tonsil fossa muscle bed. If the degree of sepsis and the appearance of the bed (bulging) indicate that there is pharyngomaxillary infection a blunt thrust through the protruding bed of the fossa will sometimes evacuate the pharyngomaxillary abscess. If this is done late in the course of an infection the possibility of parapharyngeal hemorrhage should be borne in mind, and preparation for its control by carotid ligation should have been made. If persistent bleeding follows such a procedure, ligation of the external or common carotid artery should be done. Sometimes there will be found a lead, in the form of a granulating sinus tract, connecting with the pharyngomaxillary abscess. Such a tract or lead is sometimes encountered during a tonsillectomy when performed on patients who have, at the time, no acute inflammatory symptoms or signs. If there has been bleeding from the pharynx or ear, tonsillectomy or peroral incisions of any kind are contraindicated because of the possibility of uncontrollable hemorrhage from eroded vessels. In

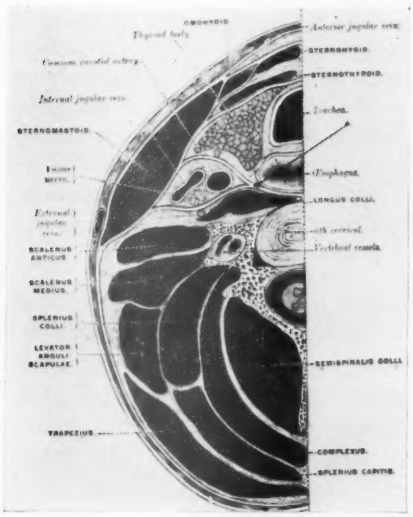

Fig. 9.—Cross section of the neck at level of the thyroid gland. Arrow points to the alar fascia. (From Gray's Anatomy, used with permission of the publishers, Lea & Febiger.)

such instances external drainage with ligation of the carotid is indicated immediately. Repeated hemorrhage after spontaneous rupture of a peritonsillar abscess may be construed as evidence of vessel erosion. Some rigidity or fixation of the neck is present in both pharyngomaxillary and peritonsillar infections. Sometimes there is torticollis toward the opposite side. It is caused by inflamed glands under the sternomastoid muscle.

Severe sepsis with spiking temperature indicates the necessity for external drainage of the pharyngomaxillary space. If sepsis persists after internal drainage has been tried, external drainage should be done. Swelling of the upper part of the neck and parotid region is usually moderate in degree. Subsidence of external neck swelling is of itself no indication of improvement. Chills are evidence of blood stream infection. If these occur repeatedly, no time should be lost in ligating or resecting the internal jugular vein and draining the pharyngomaxillary space. Even if the blood culture is negative that should be done. In the presence of severe, well-established sepsis

one well-marked chill may be regarded as evidence of blood stream infection.

Infection, with or without abscess formation, may involve both compartments of the fossa. When there is formed a large solitary abscess the prognosis is good, provided that it is at such a time reached by drainage. When the infection is cellulitic in character and there is no localization of the suppurative process, or when there are multiple small abscesses, recovery, even after drainage, is more protracted and prognosis should be more guarded.

The patient may complain of otalgia, and there is usually slight injection of the homolateral ear drum, most often limited to Shrapnell's membrane, the drum margins and the malleus handle. It is the result of congestion or inflammatory reaction in or around the auditory tube.

The foregoing description applies to pharyngomaxillary infection which is secondary to inflammation in the pharynx and tonsils. Pharyngomaxillary infection which results from infection from other parts differs in that there are no inflammatory signs in the throat. It is, however, easily recognized because there are swelling or prominence of the lateral pharyngeal wall behind the tonsil, and prominence, or displacement inward, of the tonsil and fauces which is sometimes accompanied by marked tilting of the nonedematous uvula. Trismus and parotid swelling are also present. Such infections may result from infection of the petrous part of the temporal bone which invades the parapharyngeal tissues through the inferior surface of the bone. Draining such a pharyngomaxillary infection through the submaxillary fossa approach has cured the petrous apex infection. Swelling of the parotid region may be expected in this type of case. Infection penetrating through the cortex of the mastoid tip medially causes the so-called Bezold's abscess, and may extend into and involve the pharyngomaxillary fossa. Infection from the zygomatic cells of the temporal bone causes infection adjacent to the temporomandibular joint and may point into the submandibular region, zygomatic and temporal fossae. From these regions there may be extension into the pharyngomaxillary fossa. Sudden discharge of a large quantity of pus from the external auditory canal usually signifies rupture through the canal walls. Suppurative otitis media should be ruled out, but both conditions may be present at the same time. If suppurative otitis media and mastoiditis accompany the pharyngomaxillary infection and blood stream infection occurs, the pharyngomaxillary space should be drained, the internal jugular vein should be

ligated or resected, a complete mastoidectomy should be performed, the sigmoid sinus should be inspected and, if involved, drained. In such a case the pathological condition in the mastoid should receive the same surgical treatment applicable if there were no infection of the neck present.

An abscess in the pharyngomaxillary space may rupture into the external auditory canal between the cartilaginous plates without involving the middle ear chamber. In such cases the drum presents a normal appearance with perhaps slight injection.

Infection emanating from the nasal chambers and posterior nasal sinuses is usually accompanied by some pharyngitis and is more apt to localize in the retropharyngeal lymph glands, causing abscess. This is an infection of the prevertebral region. Infection starting in one or more of the uppermost cervical vertebrae may involve the posterior compartment of the pharyngomaxillary space after penetrating the prevertebral fascia. It is usually lateral in location and is characterized by marked rigidity of the neck, marked impairment of function of the cervical spine, and perhaps torticollis or tilting toward the same side, and flexion of the neck. Radiographs of the cervical spine are very helpful for diagnosis.

It sometimes happens that infection in the pharyngomaxillary space occurs after tonsil operations, particularly when performed under local anesthesia. It is probable that in most, if not all, such instances the infection is carried in by the injecting needle. The same sequence of events may occur after injection of the mandibular nerve for dental extractions. In both instances there is involvement in the anterior compartment of the fossa, and the symptoms and signs are characteristic. Infection of the pharyngomaxillary space has followed surgical removal of the pharyngeal adenoid. Here the symptoms and signs are apt to be atypical and not frank, until, perhaps, there develops sepsis which is characterized by the typical spiked temperature curve. External drainage and perhaps ligation of the internal jugular vein should be done. Infected tonsil remnants should be looked upon with strong suspicion in the presence of deep neck infection. Such remnants should eventually be removed. Occasionally the pharyngomaxillary space is thus involved, but often the infection will involve the submaxillary or the buccopharyngeal fascia regions.

The cervical glands of the same side may be simultaneously inflamed and there may be moderate swelling. In most instances the gland inflammation is not a prominent part of the picture, but occasionally there may be localization with abscess formation, re-

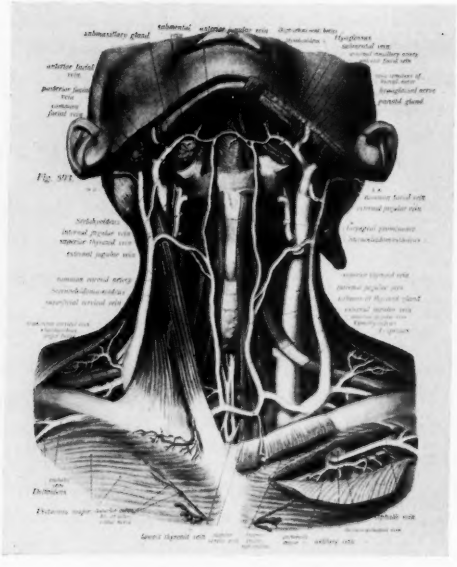

Fig. 10.—Illustration showing the rich communications of all the jugular veins with each other (anterior, external and internal). (From Sobotta used with permission of the publishers, W. B. Saunders Co.)

quiring drainage. The glands usually involved are those of the upper deep cervical chain at or near the bifurcation of the carotid artery.

The appearance of the patient varies considerably. In some instances the local signs are not frank. The patient avoids swallowing because of the accompanying pain; there may be slight or marked trismus; there may be moderate swelling of the parotid region and angle of the jaw, extending downward and forward over the adjacent upper cervical region; and there may be some torticollis toward the opposite side. If these signs are present, with prominence of the lateral pharyngeal wall and tonsil region of the same side, a pharyngomaxillary infection may be assumed to be present. With persistent general symptoms and signs of severe sepsis external drainage is indicated.

The most dangerous type of pharyngomaxillary space infection is that in which there occurs erosion of blood vessels causing ex-

travasation of blood into the parapharyngeal tissues.[9, 10] Extravasation of blood into the tissues probably is the result of erosion of arteries in most instances. Extravasation from erosion of veins is probably very rare because thrombosis usually occurs simultaneously. In the lower levels of the neck, below the digastric muscle, I have seen complete absence of the walls of the internal jugular vein, its position being outlined by a thrombus, with no extravasation of blood into the surrounding tissues. Nature provides its own cork. When veins are accidentally injured hemorrhage may be very copious until the vein is ligated, but I have yet to see copious hemorrhage from the internal jugular vein as the result of infection.

If erosion of an artery takes place slowly and there occurs an intermittent leak of small amounts of blood, there will be some clotting of the extravasated blood, sometimes in laminae. This gives rise to the so-called pseudo-aneurysm and forms a mass which causes protrusion of the lateral pharyngeal wall and which can therefore be seen. When such a blood mass, or hematoma, forces its way through the pharyngeal wall or is incised, intermittent hemorrhages of small or large amounts occur in the throat. At the same time there is no abatement of the sepsis which is severe. When pharyngeal hemorrhage occurs in a patient with a pharyngomaxillary infection the situation is indeed grave, for these initial small hemorrhages are but the forerunners of sudden massive hemorrhage which will exsanguinate the patient in a very few minutes. A patient may have the final fatal hemorrhage while in the ambulance on the way to the hospital. No matter how small the quantity of the first hemorrhage, it indicates immediate ligation of the common carotid or one of its branches and drainage of the pharyngomaxillary space by the external approach. In the performance of this surgical procedure the ligature should be placed in position around the common carotid artery, ready to be instantly tied. Then it will be safe to enter the pharyngomaxillary space. Peroral incision through the lateral pharyngeal wall is absolutely contraindicated in such cases for very obvious reasons. The hemorrhage most often occurs from erosion of the internal carotid artery, and therefore can be controlled only by ligation of the internal or the common carotid artery. I prefer to place the ligature on the common carotid artery because I believe there is less danger of acute surgical cerebral anemia. If the hemorrhage is definitely and completely controlled, the soft rubber drains elsewhere mentioned may be employed. If, however, there persists a slight or moderate oozing, gauze drains or packing are preferable.

The following is to be highly recommended in such conditions. Recently there have been evolved hemostatic materials with which I have had a limited experience in the control of hemorrhage. One is made in the form of gauze-mesh and cotton-tampon and is used locally where the bleeding occurs. Chemically it is oxidized cellulose, or cellulosic acid, a polysaccharide. It may be placed in tissues and the wound tightly sutured. It is completely absorbed. When it becomes soaked with blood it becomes black in color. It does not become a coagulum, but forms a scummy, gelatinous substance which remarkably favors the formation of thrombi in the bleeding vessels with which it makes contact. Its hemostatic effects in oozing wounds is striking. In open drainage wounds it can be removed without disturbing the thrombi which have formed in the vessels, in striking contrast with what usually occurs when ordinary gauze packing is removed from a wound.[11]

Another hemostatic substance which may be similarly employed is fibrin foam, a substance extracted from blood. It is recommended that it be moistened with sterilized human or bovine thrombin. It is stiff and brittle material and not soft and pliable. It is absorbed in the tissues.

The third hemostatic substance is gelatin sponge which is procurable under the name "Gelfoam." This is a soft, pliable, sponge-like substance consisting of gelatin which is slowly absorbable in the tissues. It is recommended that it be moistened with sterile thrombin or physiologic sodium chloride solution and lightly packed into the tissues. It has the advantage that it may be moistened with penicillin solution before being placed into an infected wound.

It is possible that the hemorrhage may come from one of the deep branches of the internal maxillary artery, in which case ligation of the external carotid artery will control it, and this is preferable to ligation of the common carotid artery. If one is not certain about the exact source of the bleeding, ligation of the common carotid offers the best chance of control.

Occasionally, when the bleeding is from the internal carotid artery, ligature of the common carotid will not control it. In such instances there is reversal of the flow of blood in the internal carotid through the circle of Willis, or possibly a reversal in the external carotid through collaterals with the external carotid of the opposite side. If such a condition exists, it will be necessary to place the ligature on the internal or external carotid, or perhaps on both. I

feel that it is probable that arterial clots form at first in the tissues ouside of the vessel and that venous clots are at first endovascular.

Mosher in his teaching has repeatedly stressed the importance of the hypoglossal nerve as a landmark in ligation of the external carotid artery. This nerve should always be identified when working in the vicinity of the carotid bifurcation. By following it backward and upward it will be seen to loop upward beneath the occipital artery, which is the first or lowest branch on the lateral aspect of the external carotid artery. This makes identification of the artery certain. The nerve crosses and lies on the sheath, is quite superficial, and may be easily injured if not recognized.

Pharyngeal hemorrhage may also be caused by necrosing, malignant, parapharyngeal new growths, but in such conditions there is no pronounced sepsis. I have observed hemorrhage in such cases. The local throat signs may, however, be similar. Such cases will be found limited to the usual cancer age period, therefore not in infants and children. Lymphosarcoma may occur in almost any age period.

The occurrence of massive pharyngeal hemorrhage in deep neck infection is one of the most frightful complications in all of surgery, comparable to the rupture of an aortic aneurysm into a bronchus or the esophagus. Frequently the initial warning is the occurrence of bleeding from the ear canal. This should be regarded as the same kind of a danger signal as the expectoration of small quantities of blood from the throat; in fact, cases with ear hemorrhage carry a high mortality.[12] Motley [10] has reported seven cases of sudden severe pharyngeal hemorrhage in deep neck infections, six of them fatal. He has compiled an excellent bibliography. Salinger's[13] report on the relative frequency of the source of hemorrhage is based on 227 cases in the literature and 10 of his own. In this series of 237 patients 76 died of hemorrhage from erosion of arteries. The internal carotid was eroded in 49, the external carotid in 4, the common carotid in 9, and other vessels in 14. Woodruff[14] has reported two cases of massive hemorrhage. Grove[15] has reported hemorrhage from an eroded ascending pharyngeal artery.

The following description of one of my cases, with hemorrhage from an eroded vessel, exemplifies the management of such cases.

A girl, six years old, No. 113716, was admitted to the hospital on January 4, 1943, with a history of sore throat of about ten days' duration and inflammatory swelling of the cervical glands on the left side of three days' duration.

A diagnosis of left peritonsillar abscess was made, and the patient was taken to the operating room for incision and drianage under general anesthesia. The

swelling involved the posterior pillar and the lateral pharyngeal wall to a greater extent than the anterior pillar. There was moderate trismus. The tonsil was not greatly inflamed or swollen. A large abscess was opened behind the tonsil after the upper pole had been freed from its attachments. In this abscess cavity there were clots of black blood.

On the evening of the following day she spat up enough blood to partly fill a small emesis basin. She was immediately taken to the operating room. The common carotid was ligated and drains were placed in the pharyngomaxillary space and on the sheath. Two lymph glands lying on the sheath were excised to afford space. Clots of black blood were found in the pharyngomaxillary space, but no free pus.

She had a severe sepsis and pronounced secondary anemia. The blood count taken after admission was as follows: red blood cells 2,760,000, hemoglobin 54%, leucocytes 23,300, polynuclears 80%, young forms 21%, eosinophiles 0%, lymphocytes 20%. While still under the anesthetic a blood transfusion of 400 cc. was given.

On January 12, 1943, the drains were removed and the patient was discharged the following day.

During her stay in the hospital she received sulfadiazine, grains 10 every four hours. The stools were black until the day before her discharge. She made a good recovery, and three months later her tonsils and adenoids were removed without incident.

CAROTID SHEATH INFECTION

In the year 1920 Mosher[16] first called attention to the relationship between deep cervical abscess and thrombosis of the internal jugular vein. Infection in the carotid sheath is secondary to infection from other layers and compartments of the cervical fascia or secondary to contacting infected cervical lymph glands. In the majority of instances it follows infection in the pharyngomaxillary fossa. Infection in the submaxillary region, of nondental origin, is also a rather frequent cause.

There are no characterizing local symptoms and signs of carotid sheath infection or internal jugular vein thrombosis. One should not expect to feel the cord-like bogginess sometimes elicited in jugular vein thrombosis caused by mastoiditis because the inflammation of contiguous tissues in the neck masks the part. Contacting lymph nodes are usually inflamed and swollen and may therefore be the reason for the pain, tenderness and swelling. If there is diffuse swelling along the course of the sternomastoid muscle, it is most often caused by inflamed glands between the muscle and the underlying vessel sheath. The only reliable symptoms are general in character and may be expressed in one word, sepsis or septicemia. The diagnosis and the time for drainage rest upon recognition of sepsis; the time for ligation or resection of the jugular vein, upon recog-

nition of infection of the blood stream. Infection along the sheath may cause torticollis toward the opposite side. Contraction of the sternomastoid muscle, induced by placing resistance against the opposite side of the chin with the hand, causes pain along the contracting sternomastoid muscle. Fluctuation may not be demonstrable even when there is abscess formation in the deep glands, because the glands are deeply situated. In such cases there may be edematous pitting on deep pressure with the finger. It causes considerable pain and the patient may object, but it is often a good sign of underlying pus. Abscess formation is not so common in the deep chain of lymph nodes as in the superficial chain. Usually the glands are the seat of well-marked inflammatory swelling. In this event it will be necessary to excise tediously some of them in order to expose the vein if ligation or resection is performed. Even when the vein is not ligated, free exposure by excision of glands brings about better drainage and tends to avoid thrombosis. Subsequent sloughing of lymph nodes, with possible secondary hemorrhage from their vessels of supply, is thus avoided. Sometimes it will be necessary to clamp small bleeding vessels in the inflamed and swollen sheath. This type of pathologic condition may be present in the absence of free visible pus.

There are four findings which, when present at the same time, definitely indicate a blood stream infection: (1) septic blood count; (2) abrupt rises and drops in temperature, so-called spiking; (3) chills and sweats; and (4) positive blood culture. If with any of these there suddenly occurs infection in a distant part, a so-called metastatic infection, one may assume that a blood stream infection is present. Of these symptoms and signs positive blood culture is the best evidence, but the least constant. Chills are not so often present in children as in adults, though severe frank chills do sometimes occur in children. Sweats, during the day or night, are often present in both children and adults. The spiking temperature is characteristic. The blood count is variable, but the percentage of young forms of the polymorphonuclears is always elevated in the presence of sepsis. While a high total white count and an increased percentage of the polymorphonuclears are expected, sometimes these phases of the count are not much over normal, thereby making it somewhat difficult to evaluate the degree of sepsis. A high young form, nonsegmented polymorphonuclear percentage is then very significant and may be considered evidence of sepsis.

Chills are almost as inconstant as the positive blood culture, especially in children. When present a chill or two is positive evi-

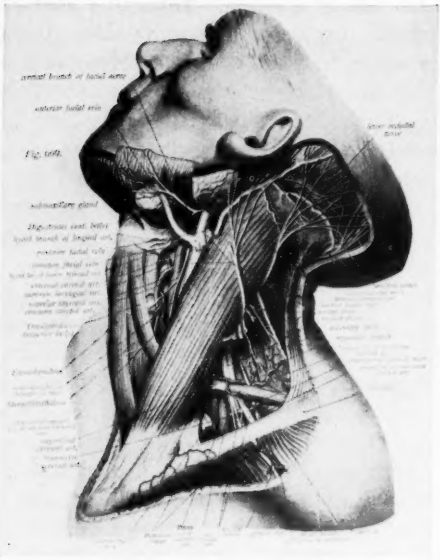

Fig. 11.—Illustration showing the relationship of the carotid sheath to the submaxillary and parotid salivary glands. (From Sobotta, McMurrich used with permission of the publishers, W. B. Saunders Co.)

dence; when absent other symptoms and signs must decide the issue. Chilly sensations may be regarded as suggestive.

Sweating, whether associated with chills or whether occurring without chills, is significant, and in the latter instance common. When sweating is profuse and frequent, dehydration and loss of salt are results which should be corrected by administration of fluids and salt in sufficient quantities. It should be borne in mind that because swallowing is painful the patient's fluid intake is apt to be considerably less than requisite, thus increasing dehydration. During the early period of the infection, before dehydration, the skin surface may be constantly moist and clammy, from sweating. Later, after dehydration has developed, the skin becomes hot and dry during the intervals between chills and sweats. When sweating is profuse and there is a marked degree of dehydration, it becomes extremely important to watch the daily urine output if a sulfonamide drug is being administered. When this drug is being taken by the patient

the excretion of urine should be at least 1000 cc. per day. Less than this amount might result in severe damage to the kidneys by crystalization of the drug. Constant intravenous fluid administration might become necessary under such circumstances. It is also important to alkalinize the urine by administration of sodium bicarbonate.

This problem of dehydration involves a full appreciation and comprehension of the problem of the water balance factors of nutrition and metabolism. The physiology of electrolyte and water exchanges is complicated and is of such extreme importance that every clinician should be familiar with the basic principles. The reader is referred to an article by Talbott[17] for authentic information on this extremely important subject. I have called freely on that article in making the following observations.

It is probably not at all uncommon for a patient to lose as much as five liters or more of fluid per day by excessive sweating in severe septic conditions. Each liter of sweat contains as much as three or four grams of sodium chloride. Unlike the kidneys, the sweat glands do not reabsorb the salts but pass them through with the sweat. Therefore the greater the quantity of sweat poured out, the greater the quantity of salt lost. Granting that all this is true it may be readily appreciated that in order to replace these losses one must be certain that water and salt are replaced through one or another of the available channels in constantly sufficient quantities, to avoid completely deficiency in the so-called fluid compartments of the body. These compartments are three in number—blood, interstitial fluid (including lymph), and intracellular fluid. It should be remembered that when there is depletion of salt the patient may complain of a thirst-like sensation after water has been swallowed in large enough quantity. This is alleviated if salt is also administered. If there is no nausea and no vomiting, water and salt tablets may be given by mouth. If there is vomiting five per cent glucose in physiological sodium chloride solution should be given by vein constantly, and not discontinued until sweating has ceased and the sepsis has diminished. As previously stated, fluid administration must not only correct the dehydration, but must also be sufficient to enable the kidneys to excrete at least one liter of urine in the 24-hour period. This is the minimum amount necessary to avoid kidney damage by the sulfonamide drugs. If kidney excretion falls below this the sulfonamide therapy must be stopped. If sulfonamide blood concentration runs high (20 mg. per 100 cc. or over), one should suspect deficient renal elimination if there are blood and

crystals in the urine in excessive amounts. Anuria is a possibility; also renal colic. Administration of adrenal cortex hormone may assist in the proper maintenance of the electrolytic salts balance.

Elman[18] states that normally about five to ten grams of sodium chloride are excreted in the urine daily, and unless this is restored an electrolyte deficit will eventually result. In the case of an adult weighing 70 kg. who shows a loss of 200 mg. per 100 cc. of serum chloride (a severe deficit) as much as 70 grams of salt would be required for its correction. In terms of physiological sodium chloride this would amount to nearly seven liters. It should not require more than 24 to 48 hours to correct this deficit. For the loss of every 100 mg. per 100 cc. of serum chloride the patient needs 0.5 grams of sodium chloride per kg. of body weight, in this case (weight 70 kg.) 35 grams, or a little over three liters of physiological sodium chloride solution.

Hypoproteinemia should be guarded against and if the patient does not take and assimilate enough food, amino acids (in the form of Amigen) should be given with the intravenous medication. The recommended solution is Amigen 5%, glucose 5% in physiological sodium chloride. Vitamins given parenterally are also advantageous. Plasma and blood transfusions are also good sources of protein.

One chill, occurring in the beginning of an acute pharyngitis or tonsillitis, does not necessarily indicate blood stream infection. It may be said that a sudden single chill of this character is caused by a shower of toxic infectious material being suddenly released into the small radicals of the venous blood system and quickly eliminated, the system being cleared in the process. The chill may be regarded as one of the manifestations of the clearing mechanism in the presence of septicemia.[19] It may be likened to an April shower. When chills appear during a well-developed infection of the neck, in which there is a continuous formation of toxins in the inflamed tissues, a definite blood stream infection may be assumed to be present. This may be likened to a prolonged storm. A constant invasion of the blood stream is believed to occur, resulting in sufficient accumulation of bacteria and toxins to produce reaction prior to their elimination. It is not believed probable that bacteria undergo much, if any, proliferation in the blood. Therefore, if checked at the source the blood stream infection should subside.

Because of this dependence on the general manifestations for diagnosis it becomes necessary to exclude other conditions which cause sepsis and other conditions in which there occurs a spiking type

of temperature. Prominent in this consideration is pyelitis which is of particular importance in female children. Mastoiditis is another possible cause of blood stream infection and may be actually coexistent; also empyema, osteomyelitis and many others. Tropical fevers, such as malaria, are characterized by rigors and high excursions of temperature, and while there would probably be no doubt in typical involvements, it should be remembered that malaria might be present in a patient with a deep neck infection. It should be constantly borne in mind that pneumonia may be present.

Definite evidence of existing or impending blood stream infection is the sudden appearance of a distant inflammation. Such manifestations are of many varieties. The following have been observed: painful, tender inflammatory swelling of the dorsum of the foot, calf of the leg, elbow, shoulder; infections in long bones, joints, the pelvic tissues, the lungs, the brain, and the mesenteric tissues. If such secondary involvements go on to abscess formation, it will be necessary to drain them surgically. Sometimes, if they occur early before surgical drainage of the neck has been done, immediate drainage of the neck may result in spontaneous subsidence of the secondary lesion. I have observed such cases. If the neck drainage is delayed or if thrombosis of the internal jugular vein occurs after the neck has been drained, it is possible that the secondary lesion will suppurate and require surgical drainage. When infection of the blood stream and jugular thrombosis are suspected or known to exist, the eye fundi should be frequently examined for choked disc, and retinal hemorrhage and thrombosis. After cavernous sinus thrombosis has taken place, the outlook is extremely serious, almost hopeless. Recently there have been reported cures in cases of cavernous sinus thrombosis following the use of heparin and the sulfonamide drugs;[20] also following the use of penicillin with and without heparin. Dicoumarin has been used with success in cases of thrombosis of the veins in the lower extremities in obstetrical conditions. These drugs, heparin and dicoumarin,[21] are active agents in retarding clotting time and thrombus formation in vivo. In the case of thrombosis in the cavernous sinus, the time for stopping the administration of anticoagulants is indicated by the recession of the papilledema. The clotting time may be markedly prolonged and thirty minutes may be considered within the bounds of safety for heparin. Heparin has the disadvantage that it must be added to a constantly flowing sodium chloride solution, given intravenously, at frequent intervals, usually every four hours. Its effect starts immediately and does not continue very long after cessation of administration.

Dicoumarin, procurable under the name "Dicumarol" has the advantage of easy administration, by mouth, one dose a day. Its effect as measured by the thrombin time in seconds is not demonstrable until after 24 to 48 hours and continues for some days after cessation of administration. No daily dose should be given before first ascertaining the thrombin time. If these drugs are used they should not be administered before surgery. Heparin may be used with penicillin by the intravenous route. If bleeding occurs blood transfusions are immediately indicated.

The most important consideration is to get drainage as near as possible to the portal of entrance, in most instances into the pharyngomaxillary space adjacent to the tonsil region. Next it is important to place drains on the widely exposed carotid sheath. If thrombosis can be immediately demonstrated, ligation or resection of the vein is immediately indicated; if not, it is probably safer to ligate if the sepsis is severe. The important necessity is to drain the original abscess or site of infection.

Penicillin is at present our best therapeutic agent for sepsis. It should be given promptly even before surgical drainage if the patient's illness is seen in an early stage, and continued after surgical drainage until the infection has subsided. The dosage should be adequate, as toxicity is practically negligible. The minimum dosage is 20,000 units every 3 hours, and when the infection is very severe this may be doubled or even trebled. Chemotherapy does not render drainage unnecessary when the indications are present and is extremely valuable as supplementary treatment to surgical drainage. In other words it should never supplant surgical drainage for abcesses. Likewise, when there is thrombosis of the internal jugular vein, ligation should be done. Administration of penicillin may be started intravenously, but because of its rapid elimination it should be continued by intramuscular injection every three hours to maintain a high blood level. Blood transfusions are indicated for anemia and for protein depletion. Plasma may also be used for hypoproteinemia.

In severe cases sulfonamides may be simultaneously administered, the patient being carefully observed for evidence of toxic effects. Chemotherapy is advancing so rapidly that what is set down in a manuscript may be out of date before it gets into print. It is not so long ago that specific convalescent and polyvalent sera and bacteriophages were strongly advocated in the early stages of an infection. I believe that they are still useful. Even the sulfonamides

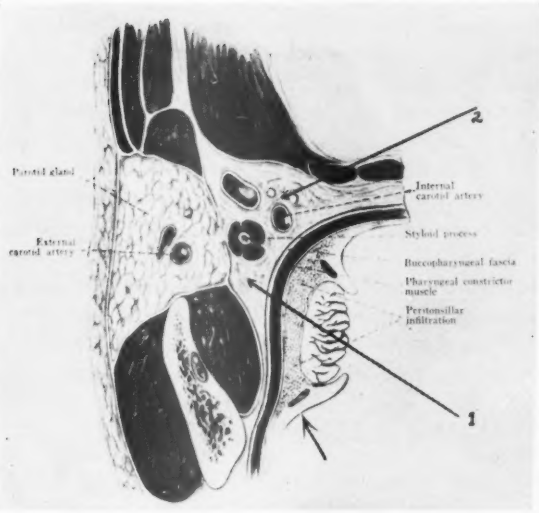

Fig. 12.—Arrow No. 1 denotes the anterior compartment of the pharyngomaxillary fossa; arrow No. 2, the posterior compartment. The illustration also shows the opening in the parotid fascia where it is adjacent to the pharyngomaxillary fossa. (From Kirschner's Operative Surgery, Ravdin Translation, used with permission of the publishers, J. B. Lippincott Co.)

now hold a secondary or minor position. When sulfonamides are administered the blood concentration and the urine should be carefully watched. The blood concentration should reach between 10 mg. and 20 mg. per 100 cc. If red blood cells or crystals appear in the urine administration should be stopped. An equal amount of sodium bicarbonate should be given, and adequate amounts of water. At least one liter of urine should be excreted daily. If sufficient amounts of fluids are not taken by mouth the venous route must be used. Dehydration must be prevented. At present sulfadiazine is the preferred sulfonamide. If penicillin or the sulfonamides are given early before abscesses have formed, subsidence of the infection often takes place. Low hemoglobin demands iron medication.

Statistics are changing constantly since the advent of chemotherapy. The figures arrived at in this text are based almost entirely on cases seen and treated before any kind of chemotherapy was available. They represent, therefore, the natural courses that events

will take without chemotherapy. It is my belief that neck infections which result from infections in soft parts yield more promptly to chemotherapy than those which result from infections in, or connected with, bony structures. For example, infections which find a portal of entrance in the tonsils and pharynx yield promptly to chemotherapy, and if abscess formation has not preceded the administration of penicillin, the infection in the neck tissues will subside without surgical drainage. Those infections which find a portal of entrance in the temporal bone or in the dental region are not so promptly controlled by the chemotherapy. If not completely controlled they may be modified to the extent that they are less fulminant, more quiescent, or even masked. As a result I have noticed a change in my original incidence statistics, and I now see a greater proportion of the Ludwig's angina, or submaxillary, type of case, and a smaller proportion of the pharyngomaxillary type of case that may require surgical drainage.

Infections of the upper respiratory tract, particularly pharyngitis and tonsillitis, are almost always promptly controlled and cured with penicillin when it is administered early. Even in later stages when there is already some peritonsillar or parapharyngeal involvement, administration of penicillin is promptly curative if there is not yet abscess formation. Gram-negative organisms may not be controlled. The fusospirochete organisms of Vincent's angina are susceptible, but the time factor may be longer.

In submaxillary region infection of dental origin penicillin is very effective, but the response proceeds more slowly. I believe that the reason for this is that bone infections (alveolar process) respond less promptly than soft part infections. I also believe that if mouths were better prepared for removal of teeth, together with preoperative administration of penicillin, there would be very few infections following dental extractions. Teeth are being extracted daily in large numbers without any preoperative preparation locally or generally. Complications in the neck will therefore be seen in about the same total numbers as in times before penicillin was available. Infection following dental extraction invades the neck rapidly, and patients do not regard it as serious until it is fairly well advanced. The average practitioner of dentistry does not yet realize the importance of early hospitalization for chemotherapy in these cases. The total incidence remains the same but the relative or proportionate incidence, formerly about 20 per cent, is much increased, probably now over 50 per cent. The general medical practitioner realizes the potential danger of an upper respiratory infection, or a so-called streptococcus

sore throat, and administers chemotherapy in an early or initial stage, thereby preventing many cases of deep neck infection. The incidence of this type, the pharyngomaxillary, the prevertebral, and the pretracheal infections, is decreasing both totally and proportionately. The relative incidence was formerly about 65 per cent. Now it is not over 40 per cent. Shortly before this was written there were under my care at the same time three patients with early pharyngomaxillary infection and one with Ludwig's angina. The last mentioned infection followed dental extraction two days previously. All four cases were controlled and cured by penicillin, the Ludwig's angina case much more slowly. All were seen in an early stage.

The importance of penicillin therapy cannot be too strongly emphasized. Those cases that are seen in well-developed and late stages sometimes pose difficult surgical and therapeutic problems. If it is evident while penicillin is being given that there is abscess formation or invasion of the blood stream, surgical intervention is definitely indicated as in days when chemotherapy was not available. Chemotherapy does not supplant surgical drainage when the indications for surgery are present. It is, however, extremely valuable in a supplementary capacity and should always be administered preoperatively and postoperatively. When penicillin is being given, the signs and symptoms of sepsis will be much less in evidence and local manifestations must be very carefully sought out. If symptoms and signs of sepsis return after administration of penicillin is discontinued, it is fairly good evidence that surgical measures are indicated. Once an abscess has formed drainage of that abscess is indicated, even though until then no complications had occurred. If improvement does not take place progressively while full doses of penicillin or sulfonamides are being administered, one should suspect the presence of an undrained abscess or a jugular vein infection which requires surgical drainage or ligation respectively. A similar conclusion may be arrived at if there is a recurrence of the infection after administration of the drug has been stopped. Antibody determination is indicated at such a time.[2] In addition to the immediate curative effects, the long-range benefits of prompt and intensive treatment with sulfonamides and antibiotics are incalculable. In these severe septic bouts, severe damage to such vital organs as the heart, liver and kidneys occurs to a greater or lesser degree. I have yet to see one escape such damage when the sepsis has been severe and prolonged. By shortening the illness and avoiding these complications, many a victim will be kept free from these dire sequelae and his life span therefore prolonged.

For dehydration three to ten liters per day, depending on the amount of sweat loss, of five per cent glucose in physiological sodium chloride solution may be required for an adult; smaller amounts are required for children. When transfusion must be delayed, for instance until a suitable donor can be found, blood plasma may be substituted for whole blood with benefit in about the same dosage and frequency. When dehydration is caused mostly by a deficient fluid intake due to nausea and vomiting there may be relatively little salt loss, and in administering fluids it is important to avoid giving an excess of salt. When the dehydration results mostly from sweating there is considerable salt loss, and it is necessary to restore this. Diaphoresis can be extreme in these septic cases.

It has been observed that there may be inflammatory involvement of the sheath and of the wall of the internal jugular vein without formation of thrombus within the vein.[22] It is my custom to resect a small segment of the vein whenever it is necessary to ligate it, because it is then possible to make a microscopic examination of the tissue. This applies to neck infection cases, but not to cases of thrombosis following mastoiditis in which ligation alone may be done. While blood stream involvement results most frequently from infection through the internal jugular vein, it should be remembered that infection may also occasionally involve the external or the anterior jugular veins. Iglauer[23] has reported suppuration within the anterior jugular vein following infection in and near the tonsil.

421 HUGUENOT STREET.

(To be concluded in the September issue.)

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Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, November 4, 1946

THE PRESIDENT, DR. FRANCIS L. LEDERER, IN THE CHAIR

Symposium

The Physiology of Speech

ITS LARYNGEAL ASPECT

HAROLD WESTLAKE, PH.D.

(Abstract)

Speech teachers have been interested in the anatomy and physiology of normal breathing, as well as in the modifications of breathing which take place during the speech act. Symptoms of certain speech disorders are described in terms of irregularities in breathing activity. In many cases speech training involves the establishment of breathing patterns which are favorable to good speech.

Presuming to teach proper breathing posed these questions. The factual bases for answers to these questions were ascertained through reasearch.

What changes take place in the thorax during breathing? The most discerning research pertinent to this point was done by Dr. Harlan Bloomer, working under the direction of Dr. Carlton Peirce in the Department of Roentgenology at the University of Michigan. The critical facts are these:

1. The essential movement for inhalation is the upward thrust of the thorax.

2. The twelve ribs move upward together in inhalation, and downward together in exhalation.

3. The first two ribs are active even in quiet respiration.

4. The vertical displacement is greatest for the upper ribs, and successively less for the lower ribs in order.

5. This latter condition emphasizes the importance of the scalenus muscles as muscles of inhalation.

6. The upward excursion of the twelfth rib in inhalation and its downward excursion in exhalation classify the quadratus lumborum as a muscle of exhalation rather than a muscle of inhalation.

7. The angle of the sternum does not change during breathing. The movement is upward and forward during inhalation, and downward and backward during exhalation.

8. The active role of the diaphragm in breathing appears to have been exaggerated. By volumetric changes, diaphragmatic shift may account for 42 per cent of the total exchange of air in the average person. Volumetric changes computed from changes in the configuration of the diaphragm cannot be entirely attributed to the specific activity of that muscle, since the widening and the upward movement of the circle of origin have their effect upon the configuration.

9. The differences between forced and quiet breathing appear to be quantitative rather than qualitative.

How does breathing for speech vary from normal breathing?

1. Varying lengths of phrases make breathing during speech less regular than normal breathing.

2. The relative durations of inhalation and exhalation are reversed. In normal breathing the inhalation phase is relatively long and the exhalation short. During speech the inhalation is short, and the phrases of speech are uttered during a much prolonged exhalation.

3. Although there is a tendency for breathing to be more rapid during speech, the average number of breaths taken by an individual in continuous speech tends to follow his normal breathing rate.

4. There is no factual basis for believing that the breathing activity during speech varies from ordinary breathing.

In considering the function of the valves at the level of the larynx during the speech act, two types of exploration have been especially productive: x-ray stills and x-ray moving pictures showing the changes in the adjustments of the cartilages as well as the soft tissues; colored moving pictures have shown the same adjustments from the superior view. The questions posed by the speech teachers, as well as the factual bases for the answers, are as follows:

Are there definite postures or relationships between the laryngeal structures during the production of specific sounds?

1. Of all the structures involved in speech production the tongue shows the greatest tendency to assume particular postures for specific sounds. The tissues of the larynx show very little consistency in their relationships for particular sounds.

Is there a manner of using the laryngeal valves which will result in a desirable vocal tone?

1. Speech teachers have described two types of vocal attack: the aspirate attack and the glottal attack. In the aspirate attack the true vocal folds approach the midline evenly, sometimes with a light contact of the vocal processes but usually they are only approximated. The exhalation appears to begin slightly before phonation begins. There is little contraction of the supraglottic tissues. In the glottal attack the vocal processes come together sharply and there is a marked tendency for the supraglottic tissues (the cushion of the epiglottis and the ventricular bands) to close in. The aspirate attack results in a desirable vocal quality; the glottal attack in a sharp or strained quality.

Can misuse of the larynx in speaking cause laryngeal irritation?

1. A study, using colored movie photography, indicated that individuals speaking with harsh voices showed notable reddening of the vocal lips after a period of continuous oral reading. The same persons, using softer, but equally loud, voices showed little or no reddening of the vocal lips during equal periods of oral reading.

2. A second study indicated that ulcerations occurring near the vocal processes of the arytenoid cartilages improved after individuals learned to use the aspirate attack and the softer voice quality described above. All the subjects used in the experiment had histories of vocal abuse during speech.

ITS AUDITORY ASPECTS

RAYMOND CARHART, PH.D.

(Abstract)

In terms of its social importance, the speech act is not completed until acoustic stimuli are heard and understood.

The evolution of speech has depended as much upon the phylogenic development of auditory capacity as it has upon increased phonatory and articulatory capacities. Similarly, we do not find in the middle ear nor in the cochlea unique developments whose presence in man accounts for his singular position as a talking animal. By contrast, we find recent structural expansions in the cerebral hemispheres (particularly in the temporal lobes, Broca's area, and the association areas) whose evolutionary developments account for human speech. As our immediate phylogenic predecessors underwent cerebral development, their capacity to utilize auditory stimuli for symbolic purposes expanded, and, in conjunction, they increasingly employed as symbols the sounds producible by the vocal mechanism. The end result was speech and language.

Any basic consideration of the auditory aspects of speech must recognize that the ability to interpret sounds as symbols for objects, qualities, actions, relations, and ideas is the keystone of human language. The present discussion is concerned with three phases of the total interpretative process: (1) the acoustic features which are the basis for differentiating the auditory patterns of speech; (2) the minimal requirements for auditory interpretation; and (3) the variables which modify these minimal patterns.

In terms of their acoustic structure, speech sounds divide themselves into six general classes. The vowel sounds plus "l", "r", "m", "n", and "ng" possess stable bands of resonance. Differentiation is based upon the portions in the frequency range at which these bands occur. Second, diphthongs and blends are characterized by a sweep from one resonance band to another. Third, frictional sounds (like "f", "s", and "sh") have broad and fluctuating concentrations of sound. Fourth, voiced fricatives (like "v" and "z") combine resonance bands with fluctuating concentrations of sound. Fifth, voiceless plosives (like "p" and "t") possess a short silent period followed by a sharp pulse of sound with wide frequency distribution and a fluctuating character. Sixth, voiced plosives (like "b" and "d")

combine the features of voiceless plosives with a low frequency component contributed by phonation.

A fact which has been recognized increasingly in recent years is that a relatively limited range of frequencies suffices for the understanding of connected speech—provided the person already has an understanding of the language being spoken. The essential frequency band for this type of hearing is from 500 to 2000 c.p.s. Loss for connected speech generally equals the average pure tone loss (in the ear under test) for the 512-2048 c.p.s. band.

The individual can ordinarily "make sense" of connected material without having to perceive all the individual speech elements, or phonemes. Listening situations exist, however, in which precise reception of the entire phonetic pattern is essential to adequate understanding. Therefore, a basic aspect of hearing for speech is phonetic discrimination. Individuals, notably those with auditory impairment, differ markedly in ability to make phonemic discriminations. This ability may be measured with special tests such as the Harvard Pb-50 lists. Deficiencies of phonemic discrimination arise from three general causes: (1) unfavorable pattern of loss (usually sharp dips in acuity for high frequencies); (2) central deficiencies in interpretative capacity; and (3) failure to learn in childhood the interpretation of auditory stimuli.

In general conclusion, the auditory aspects of speech are sufficiently complex so that he who would explore a patient's ability to hear speech must proceed with insight and care. Speech tests are the best means of exploring the patient's ability to interpret spoken language. Furthermore, these tests must be specifically selected to explore particular aspects of speech comprehension.

DISCUSSION

DR. SHERMAN SHAPIRO: I would like to have Dr. Westlake tell us a little more about the recognition of speech disorders, and I also would like an opinion from Dr. Carhart as to the scientific value of the word lists compiled by Fry and Kerridge for use in the University of London Clinics, as published in the British Medical Journals.

DR. EDWARD M. MIKKELSEN: The thought occurred to me that a more simple mechanism than the elaborate anatomic structure described could also deliver excellent results. Professional ventriloquists can dialogue several characters with complete change of voice for each with perfect enunciation. I have watched Bergen, for in-

stance, from scientific curiosity and I believe his effort to be entirely glottal.

There have been several instances of dogs trained to say a few primitive words. One I heard many years ago had been trained in Germany and pronounced German words, but he had to be prompted very much to respond, although he was very anxious to please. Many animals below the status of humans could probably learn to speak in a simple manner if they had a sufficiently high degree of cerebral development.

DR. JEROME STRAUSS: Much that has been brought out is more scientific than clinical. Yet it bears upon something that is important to all of us. I think Dr. Holinger will bear me out that we have had cases of laryngeal disease or lesion, mainly of the hypertrophic type, in which by modification of use of the larynx and without surgery the lesion disappeared. I have learned to differentiate certain of these cases but not from the standpoint of the doctor of philosophy; it is purely clinical. I have patients with malfunction of the larynx, many of whom I have classified to myself as "neckers"; that is, one can tell that they talk from the collar rather than from behind the teeth. I would like to know if there is some way in which the "neck-talkers" can modify their speech.

DR. PAUL HOLINGER: The importance of the work presented this evening has not always been understood nor appreciated in the strictly laryngological medical sense. It has always been a borderline field, sometimes associated in our minds with a pseudoscientific atmosphere that left us a little reluctant to follow. However, the work presented this evening makes us realize the firm basis this field has and the extremely thorough nature of the investigative work produced.

The discussions have been instructive from the standpoint of theory and physiology. It is fortunate that the essayists use this approach. However, we need considerably more discussions such as this, carrying the subject matter on to a presentation of pathology as well. Our interpretation of many of the concepts presented will eventually have to be in terms of laryngeal pathology, both organic and functional, as we see it in our patients. Therefore, I should like to request that we continue the discussions at another time in order to hear the opinions of those essayists on the more concrete problems we see in the functional dysphonias, and postoperatively in the benign as well as the malignant lesions.

DR. H. G. KOBRAK: I should like to ask Dr. Carhart about the practical usage of visible speech. I had the pleasure of visiting with Dr. Steinberg in New York a few months ago. He explained and demonstrated speech patterns. Dr. Steinberg compared the study of speech patterns with the learning of shorthand. A special difficulty seemed to be that the preceding sound influences the patterns. Therefore, it seems that a considerable amount of intelligence and endurance of both teacher and student is necessary to master the subject. What are the practical results so far? What place is it expected to have in the training of the deaf?

DR. HAROLD WESTLAKE (closing): How can you tell this glottal attack during phonation? I do not know that it has been proved that it can be detected accurately by the ear alone, although many speech teachers believe that they can detect it, making judgments by the sharpness of the initial tone. People who do habitually speak at levels above their optimum pitches are more apt to have nodes than those who speak at their optimum pitches or below. Those who speak below their optimum pitches are more apt to have ulceration than those who speak at their optimum pitch. In observation of the approximation of vocal folds during normal phonation, the folds appear to come together relatively evenly. In observing the approximation of vocal folds in individuals having voice disorders, one often observes a notable difference in the manner of the approximation of the folds. Frequently, the vocal processes appear to come together sharply. One often notes a constriction of the supraglottic tissue.

As to the question of neck-talkers, we are apt to introduce vague terminology when we are not sure of what is happening. Possibly a neck-talker would be making unnecessary use of extrinsic muscles. Singers, and most people who have had esthetic training, unfortunately have not had a proportionate amount of scientific training. Most studies have shown an unsatisfactory appreciation of the true structural and functional picture of the mechanism.

DR. RAYMOND CARHART (closing): I should like to discuss two questions: first, that of Dr. Shapiro regarding phonetic values. The types of tests I referred to are something quite new. Speech sounds are listed in terms of carrying power, from faintest to highest. Patients often miss certain sounds because they are not strong enough to be heard. In one kind of test we use speech materials composed of elements which carry best; with this test we measure the faintest level at which general understanding is possible. How-

ever, we can give another test in which all sounds are loud enough so that they ought to be heard with ease by the patient. We find patients who miss some speech elements even when they should be able to distinguish them. Such a test explores phonemic discrimination. Individuals vary a great deal in discriminatory capacity.

The second question is that of Dr. Kobrak regarding visible speech. Maybe we should look at the past. Visible speech represents a great victory for the Bell Telephone Company. Alexander Graham Bell tried to make speech visible, and failed. He was interested in devising something by which totally deaf persons could understand speech through sight without using lip reading. His hope was to bring the details of language to the person without auditory capacity. The Bell Telephone laboratories have now invented a method which will accomplish this end. The method is rather laborious to teach to adults, but studies made with children indicate that the latter can learn it much more rapidly than adults. The method furthermore gives us a clearer idea of what the ear must analyze, and finally it gives us a visible check on the patterns actually produced by the speech mechanism. This is an advantage in correcting speech patterns. Without the method you must rely on the residual hearing of the person or on kinesthetic stimulation. Consequently, you are limited by the speed with which you can teach new speech habits. With visible speech you can give the patient the picture and he can try to produce the correct pattern.

It is too early to say what the full value of the visible speech method will be, but reports from the University of Michigan indicate it will be very useful. We should remember, however, that visible speech supplements rather than supersedes the other methods of rehabilitation at our disposal.

Meeting of Monday, December 2, 1946

THE PRESIDENT, DR. FRANCIS L. LEDERER, IN THE CHAIR

(The Scientific Meeting was presented by members of the Staff of the Illinois Eye and Ear Infirmary.)

Posterior Nasal Hemorrhage

GEORGE WOODRUFF, M.D.

Under this heading will be included hemorrhages from the posterior portion of the nasal chambers. Postoperative and traumatic epistaxis will not be discussed. The cause of this type of bleeding appears to be hypertension or arteriosclerosis, or a combination of the two. In my experience it is limited to the middle-aged or elderly and has never been encountered in a person under the age of 40. The onset is usually sudden and the bleeding is copious, though it usually ceases when the blood pressure becomes lowered, only to recur in a few hours. This sequence usually persists until a moderate to severe secondary anemia occurs or until a condition of shock exists.

In treatment, the first requisite is to clear the nose entirely of clots and old dark blood and to remove the fresh blood as fast as it appears, so that the site of the hemorrhage may be located. Suction is very useful in attaining this goal. Anesthesia and shrinkage of the nasal mucosa is provided by the use of cocaine and adrenalin.

In my experience the bleeding area is usually found in the lateral wall of the inferior meatus, back near the choanal opening, though occasionally it comes from other areas, such as the upper posterior portion of the nasal passage.

Bleeding is usually well controlled by the use of cotton tampons, two to two and one-half inches long, coated with the precipitate of a mixture of 4% aqueous solution of tannic acid and 4% aqueous solution of antipyridine. In a typical case such a tampon is wedged firmly into the angle between the overhang of the inferior turbinate and the lateral nasal wall; three more such tampons are used to fill the inferior meatus; a fifth tampon is wedged firmly between the septum and the inferior turbinate to aid in holding the others in position.

The greatest obstacle to success appears to be a deflected nasal septum. This often prevents accurate localization of the bleeding area and prevents proper placement of the tampons. In some such cases a choanal plug, pulled forward from the nasopharynx and wedged tightly into the posterior nares, may prove successful. Gauze strips may then be packed against it from the anterior nares. When bleeding has not been controlled by packing, the hemorrhage has been arrested by ligation of the external carotid; this should be carried out before the loss of blood becomes extreme.

When indicated, lost blood should be replaced by transfusion.

Routine examinations with the nasopharyngoscope have revealed the presence of a group or plexus of blood vessels running from the posterior part of the inferior meatus downward and backward into the nasopharynx. In many older people these vessels are definitely dilated and they appear to be the probable source of the hemorrhages that frequently occur in this area. The name naso-nasopharyngeal plexus is suggested for this group of vessels.

DISCUSSION

DR. O. E. VAN ALYEA: While discussing the subject of nasal hemorrhage aside from that from the area of Kiesselbach, we should not lose sight of the fact that hemorrhages occur in the antrum. Some years ago an article by Hall and Thomas appeared in the Archives of Otolaryngology, in which a series of cases of hemorrhage from the antrum was reported. The theory was that sudden sneezing or violent nose-blowing ruptured a vessel in the mucosal lining of the cavity. It is well, in cases of nasal hemorrhage of undetermined origin, to transilluminate or x-ray the sinuses; clots in the antrum may be revealed. The authors originally treated such cases by a Caldwell-Luc procedure; later they tried irrigation only and found that this removed the clots and stopped the bleeding. I have used that method successfully in several cases. I would like to reiterate that this area should not be overlooked as a possible source of nasal hemorrhage.

DR. E. J. BLONDER: Hemorrhage from the posterior part of the nose may often be controlled by injection about the bleeding area with a solution of 1% novocain, or physiologic sodium chloride solution, using a fine needle. Blood and clots may refill the nose before a cautery can be applied. A number of years ago I started injecting novocain solution for the control of such hemorrhages. After the bleeding has been controlled, the operator has an opportun-

ity to view the ruptured blood vessel and cauterize it if he desires to do so. I seldom use cautery, because if the bleeding is due to a sclerosed blood vessel bleeding may recur from the margins of the cauterized area. Powdered sugar, either as a temporary pack or by instillation with a powder blower, has some merit. Another excellent method is to inject a sclerosing solution about the blood vessel with a long fine needle. As a rule I use no pack in treating these hemorrhages.

Dr. Maurice F. Snitman: Has Dr. Woodruff been able to determine whether the nasal bleeding is venous or arterial? Would ligation of the external carotid artery be of value in cases in which the bleeding is venous? Personally, I do not think so, since venous anastomoses with the surrounding regions are of larger caliber and more abundant than the corresponding arterial supply.

Dr. Robert Henner: Several new products that act as hemostatic agents have been found useful in the control of severe epistaxis. I have had experience with the fibrin foam pack and the gelatin sponge. Both these materials are soaked in thrombin prior to application. The latter has more body to it for manipulation; whereas the former has pliability for insertion under the inferior turbinate. On one occasion a patient with severe hypertension who was bleeding profusely resisted all other techniques for stopping a posterior bleeding vessel such as Dr. Woodruff described. Direct application of a thrombin-soaked fibrin foam pack immediately controlled the hemorrhage. The pack is absorbed and therefore does not require removal. Complete visualization is not necessary, but the pack must be placed in direct contact with the bleeding point.

Dr. Alfred Lewy: I have heard of an interesting experience in stopping bleeding by using oxidized cellulose, and I would like to hear from Dr. Woodruff on this.

Dr. Thomas C. Galloway: After trying irradiation and diathermy without much success in bleeding from hereditary telangiectasis, we have recently been able to control this well from such areas by injection of sclerosing solutions of quinine and urethane or sylnasol.

Dr. George Woodruff (closing): So far as I know, a spontaneous hemorrhage of the antrum did not occur in any of my cases. However, it may have been overlooked and in any event I know of no reason why it could not occur. Novocain injections may be of value in controlling nasal hemorrhage, but in these copious hemor-

rhages deep in the posterior portions of the nose I am somewhat doubtful of their value. Theoretically it would be a good thing to have suture material tied to the tampons and fastened on the face. However, it complicates the packing and with the technique used none have so far slipped backward.

Ligation of the external carotid has been successful in all four of the cases in which it has been used. Perhaps this is an argument that the hemorrhages were all arterial, but I am not sure. I have had no experience with ligation of the internal maxillary artery, but it seems that it would be a more difficult procedure than ligation of the external carotid artery.

Nasal Melanoma

ALFRED LEWY, M.D.

This man, a negro, aged 44, first came to the Infirmary in October 1944. We found a dark bluish tumor filling the left side of the nose. The x-ray film showed the left maxillary sinus to be cloudy because of thickening; the other sinuses were negative. A biopsy was done, and the pathologist's report stated definitely that it was a melanoma. It appeared to be about the consistency of an ordinary nasal polyp and was apparently attached to the septum. All that part of the septum, including the base of the tumor, was removed by a cautery knife—practically the entire cartilage of the septum and perhaps some of the bone; we do not know just how far we went because the operation was performed with the electrocautery knife.

After a few months there was a recurrence, the tumor being almost as large as the original one. The incision was widened, and this time there was an invaded area at the junction of the septum with the ridge in the nose, all of which tissue was removed. Subsequently there were three recurrences, each under the ridge of the nose where the septum would have joined had there been any septum left. Each time the growth was destroyed by the electric needle. It has now been several months since we have found any trace of the melanoma.

Melanomas in this area are exceedingly rare, and they are especially rare in negroes. McCullen says it is a curious fact that in horses melanomas practically never occur in any but white or gray horses, and that all melanomas observed at autopsy have occurred in white persons.

We are having the patient return at frequent intervals for observation. We have found nothing but a small depression of the ridge of the nose. There is a spot on the forehead which resembles a melanoma, but the patient says it has been there for as long as he can remember. This has been left untouched.

An Unusual Laryngeal Tumor In An Aged Man

ALFRED LEWY, M.D.

J. N. V., a man aged 81, entered the Eye and Ear Infirmary on February 16, 1945, complaining of hoarseness and respiratory obstruction. On examination a round, whitish, smooth, shining neoplasm was seen occupying the introitus of the larynx, so that nothing could be seen below it. Under local anesthesia the growth was easily divulsed under direct laryngoscopy, leaving a smooth raw surface on the left side of the larynx above the cords. The patient left the hospital on February 19, 1945, in good condition. The tumor was round, had a smooth, shining capsule, and was clinically consistent with a fibroma, which was also the first histologic report.

The patient returned on July 19, 1945, with a recurrent growth obstructing the larynx. This time it appeared as a grayish irregular mass. It was removed under local anesthesia by laryngofissure, and a median tracheotomy was done. Sections from this tumor were diagnosed by several well-known pathologists as a fibrosarcoma.

In spite of his age, the patient showed no ill effects and left the hospital within a few days. He was sent to Illinois Research Hospital for irradiation therapy, returning to the Infirmary at intervals for observation. The last time he was seen before his demise he had removed the tracheal tube, the tracheostoma had nearly closed and there was no evidence of recurrence, so another tracheotomy was not performed.

The second tumor measured 3 x 13 cm. It was attached at the original site and hung down into the trachea. It is remarkable that the patient could breathe as well as he did.

On January 3, 1946, the patient was brought to the hospital in extremis and died before another tube could be inserted. The interesting features in this case are the age of the patient, the size of the tumor, the mistaken original diagnosis and the mistaken judgment in permitting the tracheotomy wound to close.

DISCUSSION

DR. MAURICE F. SNITMAN: There are a number of cases of fibroma in which the determination of a sarcomatous quality is difficult. Furthermore, in the same specimen some areas are definitely more malignant than others; accordingly, further sections of the biopsy material may be of assistance in establishing a more accurate diagnosis. Because of the poor response of fibrosarcomas to irradiation, surgery is usually the proper method of treatment. It would be inadvisable to employ postoperative irradiation; such treatment might prevent further surgical measures to a local recurrence.

DR. ALFRED LEWY (closing): The biopsy of the first tumor was studied extensively. After a long search two pathologists stated that some malignant cells were found in the outlying area. If they were there, they were overlooked at first.

Ear, Nose and Throat Survey of Children in the School for the Deaf At Jacksonville, Illinois.

M. TAMARI, M.D.

AND

R. SPEAS, M.D.

(This article appears in full on page 425)

DISCUSSION

DR. SHERMAN SHAPIRO: I want to say a few words about the diagnostic problem in these cases. Although I happen to be a "tuning fork" man, I believe that in a study of this type the audiometer will be of more value than tuning forks; particularly, a sweep-range type of machine would be invaluable for discerning islands of hearing. Tuning forks can, of course, be used also, but only according to a standardized technique such as the one advocated by the Committee on Tuning Forks appointed by the Academy of Ophthalmology and Otolaryngology a number of years ago.

Abstracts of Current Articles

EAR

Effect of Obliteration of the Endolymphatic Sac and Duct in the Monkey.

Lindsay, J. R.: Arch. Otolaryng. 45:1-13 (Jan.) 1947.

The author operated on seven monkeys' ears, attempting to destroy the endolymphatic sac and duct. None of the animals showed postoperative disturbance of equilibrium or any nystagmus.

The extent of the operative lesion was determined by histologic examination.

In three ears not more than exposure and possibly simple drainage had been accomplished. In the other four, the sac and duct were largely destroyed.

These experiments demonstrate that a normal quantity of endolymph is not dependent on the existence of the endolymphatic sac or the more differentiated medial dilated portion of the duct. The author believes that obliteration of the endolymphatic sac to this extent without injury to the remainder of the system rules out the hypothesis that hydrops of the labyrinth is due to impairment of the function of this structure.

HILDING.

Sound Location and Its Determination in Connection with Some Cases of Severely Impaired Function of the Vestibular Labyrinth but with Normal Hearing.

Diamant, H.: Acta Otolaryngologica 34:576, 1946.

From a review of the literature and a study of three cases of his own, the author concludes that the theories advanced by Guttich that the vestibular apparatus plays a dominant part in the location of sound are without foundation. His own findings show that persons with impaired or abolished vestibular function are able to localize sound as well as those with normal vestibular function.

HILL.

Testing the Hearing of Newborn Infants.

Froeschels, Emil, and Beebe, Helen: Arch. Otolaryng. 44:710-714 (Dec.) 1946.

A study of the hearing of newborn infants was made on a group of 33, varying from one-half to nine days old. Urbantschitsch's whistles were used in the testing, since tuning forks were found to be unsatisfactory. Blinking was found to be the most frequent reaction. Jerking movements and shrinking or turning toward the source of sound were less frequent reactions. Of the 33 infants, 31 reacted positively, thus proving the presence of hearing in the majority of newborn infants.

HILDING.

New Treatment for Hearing Disorders

Hirschfeld, Hans, et al: Arch. Otolaryng. 44:686-700 (Dec.) 1946.

This paper describes a treatment for hearing disorders consisting of intramuscular and oral administration of a combination of amino acids and vitamins. Seventy-eight patients were treated of whom 57 showed improvement. The treatment seems to be effective for the nerve type of deafness. The high tone perception improved.

HILDING.

Diseases of the Ear Due to Louse-Borne Typhus.

Merei, J.: Acta Otolaryngologica 34:572, 1942.

In a series of 800 cases of typhus fever studied by the author, acute otitis media was not a common finding. Involvement of the internal ear was noted in 60 to 65 per cent of cases. Marked tinnitus and hearing impairment of the perceptive type together with vertigo were so common as to be considered an early diagnostic sign. Hearing gradually improved by the twenty-fifth day but the vestibular involvement lasted several weeks.

HILL.

On the Treatment of Apicitis Suppurativa.

Thornval, A.: Acta Otolaryngologica 34:546, 1946.

In a report of three cases of apex suppuration the author recommends the employment of a modified Streit operation, feeling that when carefully performed the risks can be considerably min-

imized and that it has the advantage of ease and simplicity of execution. The dura of the posterior fossa was not exposed nor was the posterior buttress removed. Dura was carefully lifted from the superior surface of the petrous until the dural fold containing the gasserian ganglion was reached. Here the bone was perforated and the abscess cavity evacuated by suction.

This technique would seem applicable only to cases definitely localized at the apex.

HILL.

NOSE

The Connection Between Osteomyelitis of the Cranial Bones and Inflammatory Processes in the Nose and Its Accessory Sinuses.

De Kleyn, A.: Acta Otolaryngologica 34:534, 1946.

In a general discussion of osteomyelitis of the cranial bones the author pleads for correct individualization in therapy rather than following any set routine. The virulence of the bacteria and the individual susceptibility of the patient are of more importance than the nature of the bacteria. Metastatic osteomyelitis can be localized in various parts of the cranium while the primary focus may be difficult to find even at autopsy. A case is reported in which a frontal lobe abscess secondary to osteomyelitis and frontal sinusitis was believed to be metastatic from a large liver abscess.

HILL.

Improved Surgical Technic Based on Modifications of Jansen-Ritter and Caldwell-Luc Procedures for Chronic Sinusitis.

Gatewood, W. L.: Arch Otolaryng. 45:14-39 (Jan.) 1947.

The author reviews the anatomy of the paranasal sinuses and describes an external operation for removal of the lining of the frontal sinus and exenteration of the ethmoids and opening of the sphenoid. In the case of the sphenoid, he curettes only the medial and inferior walls because of the danger involved in disturbing the superior, posterior, and lateral walls. The operation upon the maxillary seems to be the standard Caldwell-Luc with mucous membrane flaps made from the nasal lining of the inferior meatus.

(One wonders if it is possible to be sure of removing all of the mucous membrane in the frontal sinus in the manner described. W.

Likely Simpson has advocated the use of malleable curettes together with mirrors for removing this membrane. The treatment of the sphenoid might also be open to question. Ed.)

HILDING.

PHARYNX

Penicillin in Peritonsillar Abscess and Peritonsillar Phlegmon.

Rosenberg, Abraham: Arch. Otolaryng. 44:662-667 (Dec.) 1946.

This study reports 25 cases of peritonsillar abscess and phlegmon treated with penicillin. The routine dosage was 200,000 units given intramuscularly in ten doses at three-hour intervals. The author found penicillin to be superior to sulphonamides in treatment. If abscess had already formed, he found it necessary to incise and drain it. Penicillin seemed to have a definite curative action in phlegmon and tended to prevent abscess formation.

HILDING.

Peritonsillar Abscess and Mediastinitis.

Richtner, N. G.: Acta Otolaryngologica 34:521, 1946.

Mediastinitis is more apt to be a complication of retropharyngeal abscess than of abscess in the tonsillar region. In the latter case infection may extend into the parapharyngeal space with involvement of the great vessels, before downward extension to the mediastinum. However, when mediastinal infection occurs secondarily to tonsillar phlegmon, it is usually very malignant and fulminating. The author reports a case in which the symptoms led to an exploratory laparotomy for suspected ruptured duodenal ulcer and in which autopsy revealed mediastinal involvement extending to the diaphragm.

HILL.

The Treatment of Residual Lymphoid Tissue in the Nasopharynx by Radium.

Gay, Leslie N.: J. Allergy 17:348-351 (Nov.) 1946.

The author after describing the work at Johns Hopkins University relative to the application of radium for the elimination of pharyngeal lymph tissue responsible for defective hearing points out the importance of surgical removal of any very large masses of adenoid

tissue. It has been demonstrated that the size of the lymphoid tissue is not so important as the location. In or around the tubal orifices it does the greatest amount of damage. There is a definite relationship between tonsil and adenoid infection and bronchial asthma. It was also found that many of the children deaf for the higher tones were sufferers of bronchial asthma. These also had a history of recurring colds, almost invariably associated with asthmatic attacks. Good results were also obtained with the radium treatment in the prevention of these recurrent colds and asthmatic attacks. A persistent cough, due to postnasal drip, was dramatically relieved in many instances.

The author strongly advises a careful nasopharyngoscopic study in all children with recurrent colds, asthma and any evidence of deafness, even though it be in the upper tones.

McLAURIN.

ESOPHAGUS

A Case of Fatal Lesion of the Mucous Membrane of the Esophagus Feigning a Perforating Peritonitis.

Kecht, B.: Acta Otolaryngologica 34:587, 1946.

The author reports a case of periesophageal abscess and purulent mediastinitis, mistakenly diagnosed as a perforated duodenal ulcer. Autopsy revealed a small splinter of wood which had punctured the esophageal mucous membrane and lodged in the submucous tissue. Symptoms of peritonitis immediately after swallowing the splinter led to abdominal exploration. An apparently superficial injury of the esophagus led to a chain of serious sequelae with empyema, gangrenous pneumothorax, collapse of the lung and death.

HILL.

MISCELLANEOUS

The Question of the Etiology and Treatment of What Is Known as Rheumatic Facial Paralysis.

Bergström, E.: Acta-Otolaryngologica 34:541, 1946.

The author describes a case of 15 years' duration which cleared after decompression operation. In this case the nerve appeared normal from the stylomastoid foramen to just below the horizontal

semicircular canal. At this point the facial canal was found contracted to about one-half its former size and the nerve compressed. Exposure was continued upwards, sacrificing the middle ear with the usual radical mastoidectomy technique. Normal appearing nerve was found just before it entered the pyramid. Recovery was practically complete in seven months. The author feels that if there are no signs of improvement in cases of rheumatic facial paralysis (Bell's palsy) in four months operation should be considered.

HILL.

Disturbances of the Ethmoid Branches of the Ophthalmic Nerve

Littel, J. J.: Arch. Otolaryng. 43:481-499 (May) 1946.

The author describes a syndrome due to pressure and obstruction in the olfactory fissure. It includes:

1. Pain or aching in or about the eye which is relieved by application of cocaine and privine on a swab.

2. Nasal stuffiness, worse on the affected side (sometimes so slight that the patient is unaware of it).

3. Postnasal discharge, perhaps evidenced only by an inflamed lateral pharyngeal band.

4. Systemic symptoms of absorption of a toxin.

The pressure and obstruction in the olfactory fissure are due to thickening of this part of the septum and perhaps undue prominence of the ethmoid cells and middle turbinate. The pain is due to disturbance of the anterior ethmoid nerve, resulting from inflammatory changes in the obstructed olfactory fissure. The entire syndrome can be relieved by submucous resection alone or together with ethmoidectomy. It can also be relieved by section of the anterior ethmoid nerve through an external operation.

HILDING.

Parotidectomy: Indications and Results.

Bailey, Hamilton: Brit. M. J. pp. 404-407, March 29, 1947.

Because of the fear of producing a facial paralysis the term superficial lobectomy is a more desirable term for most surgeons than subtotal parotidectomy and it will probably remain so until it is

conceded that the classical descriptions of the course of the facial nerve are inaccurate. Furthermore the profession at large fails to recongnize the fact that it is only a matter of time before the facial nerve will become involved by a tumor of the parotid with or without surgery.

Joseph McFarland, the pathologist, pointed out that the recurrence rate was much higher after the removal of small tumors than after the removal of large ones. Because of this teaching the pernicious habit of waiting until the tumor became larger came into being. Furthermore Bailey finds it hard to determine, preoperatively, whether a respective tumor is small or large. If the tumor arises deep in the gland, it may reach considerable size before it produces a large external swelling. McFarland remarked, "I have sometimes seen little tumors the size of peas or pinheads in close juxtaposition to the larger easily recognized tumors." Bailey holds that this is the explanation of the frequency of recurrences and constitutes a strong argument for subtotal or total extirpation of the parotid gland in mixed tumors of the parotid.

Instead of the usual small transverse incision used for fear of injury to branches of the facial nerve, Bailey uses a J-shaped incision beginning at the level of the zygomatic arch, running down just anterior to the pinna and curving around the root of the lobule to end on the tip of the mastoid. A large flap of skin is reflected by undercutting. No attempt to remove any parotid tumor should be made until the whole gland is in full view. Ligation of the external carotid reduces bleeding. The temperofacial divisions of the facial nerve are clearly seen as soon as the superficial lobe has been completely mobilized. Sufficient parotid tissue should be removed to ensure that the tumor capsule has not been encroached upon. Bailey discovered that it was possible to resect large portions of the parotid gland without the development of salivary fistula. He had only one such fistula in a series of 66 operations which included 33 total or subtotal gland resections.

Bailey states that the parotid gland is a bilobed structure consisting of a large superficial and a small deep lobe connected by an isthmus. Theoretically, a neoplasm can arise from any portion of the gland but most mixed parotid tumors begin in a comparatively small area a little in front of and above the angle of the jaw. Another starting point but less common than the foregoing is in the region just anterior to the tragus. The differential diagnosis between such a tumor and a preauricular gland is sometimes very difficult.

Sometimes the mixed tumor becomes cystic and the assumption that such a cyst is innocuous is erroneous. True cysts of the parotid are extremely rare. In such an instance sialography will indicate blockage of a branch of the parotid duct tree by a stone. Cystic adenolymphomata which arise from lymphoid elements around the first branchial cleft are usually benign but are exceedingly rare. They are hard to diagnose from degenerating mixed parotid tumors.

All mixed parotid tumors are completely radioresistant and irradiation is without the slightest benefit on parotid tumors except in the very exceptional case of an adenolymphoma.

After operation on the parotid gland that involves dissection of the seventh nerve a major degree of facial palsy always occurs and may persist for weeks or months. In four cases in which Bailey knew he had cut a primary division of the facial nerve recovery took place completely or to such a degree that the patients were unaware of any loss of function. Since studying the description of the facial nerve by McCormack, Cauldwell and Anson, Bailey has verified on the living the anastomotic twigs connecting the temperofacial and the cervicofacial divisions of the seventh nerve anterior to the parotid isthmus. He believes that it is the existence of these anastomotic communications that accounts for the eventual restitution of function of the nerve when one of its primary branches is severed.

Perhaps what is more to be feared when nerves in and around the parotid gland have been damaged is not the muscle palsy but the much less known auriculotemporal syndrome of Frey in which, when the patient eats, the cheek becomes red, hot and painful and perspiration appears on it. This is also associated with a hyperesthesia of the face.

—GROVE.

Allergic Parotitis.

Waldbott, G. L., and Shea, J. J.: J. Allergy 18:51-54 (an.) 1947.

Waldbott and Shea have reviewed the literature and found that a good many cases of allergic parotitis have been reported. The first case that they found reported was one that appeared in the literature in 1879 and in practically all of these cases there were other allergic manifestations. In other words, parotitis was just one of the allergic reactions that each patient suffered.

They call attention to the fact that Pearson reported 13 cases of recurrent parotid swelling that he classed as allergic, and that in all of these cases there were other allergic manifestations such as asthma, urticaria, hay fever, spasmodic rhinorrhea, angioneurotic edema, eczema and abdominal colic after ingestion of certain foods. There was also a familial history of allergy in six of these cases.

The authors report three new cases and have very carefully investigated each case from an allergic standpoint. They have proven allergies to exist in some form in each of the three.

One of the most important comments that these authors make is that they believe that the swelling of the parotid gland is due primarily to an allergic edema of the mucous membrane lining the duct and that it is similar to that seen in the bronchi in the presence of allergic asthma. They also feel that there is a possibility that there may be a primary allergic edema of the parotid gland itself but that this is not so likely to explain the glandular swelling as is the more probable allergic reaction that occurs in the mucous membrane lining the duct. They summarize their paper as follows:

"1. Three cases of recurrent parotid swellings in asthmatics are presented. They usually occurred shortly before the onset of asthmatic seizures. In one of these patients, the swellings had been present for many years before the development of asthma.

"2. Foods were definitely identified with the production of these attacks, and their elimination produced relief.

"3. In view of this observation, allergy should be considered as a factor in the production of certain cases of recurrent parotitis."

McLAURIN.

New Methods for the Control of Air-Borne Infection with Special Reference to the Use of Triethylene Glycol Vapor.

Robertson, O. H.: Wisconsin M. J. 46:311-317 (March) 1947.

For the disinfection of air, triethylene glycol has been found to be the most suitable. When dispersed into the air in vapor form it is odorless, tasteless, nonirritating, nontoxic, invisible and requires extremely small quantities to produce a bactericidal atmosphere. When tested under experimental conditions, concentrations of the

glycol as low as 1 gm. dispersed in one-half billion cc. of air produce rapid death of the various respiratory pathogens in the air. A room 10x10x8 feet requires the vaporization of one drop of glycol. It has no deleterious effect on walls, fabrics, books or other objects.

Under optimum conditions consisting of a temperature of 70 to 80 degrees F., a relative humidity of under 60 per cent and a quantity of glycol vapor sufficient to produce at least half saturation of the air, the rate at which such organisms as the streptococcus hemolyticus are destroyed is very rapid. The pneumococcus is even more sensitive than the streptococcus while staphylococci are slightly more resistant.

Adequate atmosphere humidity is essential for proper bactericidal action as the glycol vapor has little effect upon dried bacterial particles floating on the air. As 50 to 70 per cent of the glycol vapor dispersed into the air is rapidly lost by condensation on surfaces and air-borne dust particles, the vaporization of the glycol must be continuous. The vapor must be distributed evenly throughout the treated space. As it has very little tendency to diffuse itself, air currents produced by electric fans or some other means must be generated. A special apparatus for regulating accurately the concentration of the glycol vapor in the air has been devised by Puck, Wise and Robertson.

Triethylene glycol vapor has been used in hospital wards, military barracks and industrial establishments.

GROVE.

Penicillin Therapy in Otorhinolaryngologic Practice.

Krass, F.: Arch. Otolaryng. 44:647-661 (Dec.) 1946.

The author reports a study on 113 cases of upper respiratory infection including otitis media, sinusitis, and mastoiditis. The sodium penicillin was used by instillation and by the Proetz displacement method as well as by parenteral administration. Topical application in acute cases both in ears and sinuses seems to have definite value but is more effective when combined with parenteral administration. Nose drops containing penicillin are of doubtful value. The value of penicillin in chronic otitis media is also doubtful.

HILDING.

The Prediction of Constitutional Reactions by Testing with Ragweed Pollen Fractions.

Zonis, Jonathan, and Rubin, Nathan.: *J. Allergy* 18:36-38 (Jan.) 1947.

These two authors call attention to the efforts that have been made by a number of allergists to determine whether it was possible to predict certain constitutional reactions by testing with ragweed pollen fractions. The first report of work of this nature was done in 1942 by Drs. Sherman and Hebald, who felt that there was a definite difference in the reactions produced by the No. 1 and No. 2 ragweed fractions and concluded that by proper testing with these two fractions it was possible to determine in advance which patients were more apt to have constitutional reactions and therefore should be treated with special caution.

Later, in 1944, Cooke reported his investigations along this same line and came to the conclusion that patients very sensitive to fraction No. 2 did not tolerate large doses of ragweed and that they should be handled with special caution.

The authors have carefully prepared the two fractions of short ragweed extract, determining No. 1 as being the precipitate from 0 to 50 per cent saturation with ammonium sulfate, and the second one the precipitate from 50 to 68 per cent saturation with ammonium sulfate. After running tests on 50 patients they came to the following conclusions:

"Since our figures show no significant disproportion between the two groups of patients, and since none of our patients reacted to one fraction alone, we are forced to conclude that in our hands these ragweed fractions were of no value in determining a possible constitutional reaction in a ragweed sensitive patient during therapy. Our results are best explained on the basis that ragweed fractions are not pure chemical substances, as pointed out repeatedly by Cooke and others. We believe that our fractions are as nearly pure as can be obtained by fractionation with ammonium sulfate."

McLAURIN.

Treatment of Intractable Asthma.

Barach, Alvan L.: *J. Allergy* 17:352-357 (Nov.) 1946.

Barach emphasizes the importance of continually making an effort to determine the allergic factor that may be responsible for the maintenance of persistent bronchial asthma. In addition to

adopting measures of desensitization or the avoidance of definite allergens, the author has reviewed certain therapeutic measures that have proven of considerable value in giving relief to these patients.

In the more intractable cases of asthma, Barach advises the administration of oxygen-enriched atmospheres in conjunction with demerol and possibly the addition of some vasoconstrictor such as aminophyllin.

He warns against the use of large doses of demerol because of the possibility of nausea, dizziness and vomiting. He has also gotten very good results by adding 1/150 to 1/100 grain of atropine to the aminophyllin that is administered two or three times daily, and prefers this type of medication to adrenalin.

Intermittent inhalations of 80 per cent helium and 20 per cent oxygen mixtures have been employed in some cases with good results in bringing about a relief of their dyspnea. He also advises the use of ether anesthesia to give relief in some of the most intractable cases and explains it is best given rectally in dosages of 80 to 90 cc. in equal amounts of olive oil over a period of 30 to 40 minutes. This may have to be repeated for two or three consecutive days.

The author has been using penicillin aerosol for the treatment of the bronchial infection that accompanies many of these cases and thinks he obtains the greatest relief in those cases in which a hemolytic streptococcus or a pneumococcus infection exists, and less satisfactory results when streptococcus viridans is the predominating organism. Barach is under the impression that chronic sinus disease with exacerbations plays a considerable part in the production of these very severe asthmas and is convinced of the value in clearing up these sinus infections if possible.

Finally, it may be stated that a combined therapeutic program may be necessary in patients who are sensitive to a multiplicity of substances. This program may include physiologic and antibiotic therapy, diet, the use of the antihistamine drugs, and all the efforts possible to desensitize the patient against the allergens.

McLAURIN.

Notices

AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology will conduct examinations at the Palmer House, Chicago, Illinois, October 7 to 11, 1947. Communications should be addressed to the Secretary, Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.

HOME STUDY COURSES

The Home Study Courses sponsored by the American Academy of Ophthalmology and Otolaryngology, in the basic sciences of those two specialties, will be given again beginning Sepetmber 1, 1947. Registrations must be completed before August 15. Detailed information may be secured from Dr. William L. Benedict, Executive Secretary, 100 First Avenue Building, Rochester, Minnesota.

UNIVERSITY OF CALIFORNIA

The University of California Medical School announces the following postgraduate course in otorhinolaryngology to be given September 8 to 12, inclusive, 1947. Requests for information and for registration should be addressed to Dr. Stacy R. Mettier, Head of Postgraduate Instruction, Medical Extension, University of California Medical Center, San Francisco, 22, Calif.

OFFICERS

OF THE

NATIONAL OTOLARYNGOLOGICAL SOCIETIES

American Academy of Ophthalmology and Otolaryngology

President: Dr. Alan C. Woods, Johns Hopkins Hospital, Baltimore 5, Md.
President-Elect: Dr. Carl H. McCaskey, 608 Guaranty Bldg., Indianapolis, 4, Ind.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., October 12-17, 1947.

American Broncho-Esophagological Association

President: Dr. Robert M. Lukens, 1001 Medical Arts Bldg., Philadelphia 2, Pa.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago 11, Ill.

American Laryngological Association

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis 8, Mo.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Meeting: The Homestead, Virginia, Hot Springs, April 14, 15, 1948.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Lyman G. Richards, 1101 Beacon St., Brookline, Mass.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Meeting: Chalfonte Haddon Hall, Atlantic City, N. J., April 7, 8, 9, 1948.

Sections:

Eastern—Chairman: Dr. J. Winston Fowlkes, 12 East 63rd St., New York 21, N. Y.
Southern—Chairman: Dr. William A. Wagner, American Bank Bldg., New Orleans 12, La.
Middle—Chairman: Dr. Hugh G. Beatty, 150 E. Broad St., Columbus, Ohio.
Western—Chairman: Dr. Meade Mohun, 23 Second Ave., San Mateo, Calif.

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles 14, Calif.
Secretary: Dr. Fletcher D. Woodward, 104 E. Market St., Charlottesville, Va.
Meeting: Atlantic City, N. J., June 9-13, 1947.

American Otological Society

President: Dr. Bernard J. McMahon, 634 N. Grand Ave., St. Louis 3, Mo.
Secretary: Dr. Gordon D. Hoople, Medical Arts Bldg., Syracuse 3, N. Y.
Meeting: The Homestead, Virginia, Hot Springs, April 12, 13, 1948.